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Science and Practice of Medicine.

VOLUME I.

THE
SCIENCE AND PRACTICE
OF
MEDICINE.

BY
WILLIAM AITKEN, M.D., EDIN.,
PROFESSOR OF PATHOLOGY IN THE ARMY MEDICAL SCHOOL.

SECOND AMERICAN, FROM THE FIFTH, ENLARGED AND CAREFULLY
REVISED, LONDON EDITION,

ADOPTING THE NEW NOMENCLATURE OF THE ROYAL COLLEGE OF PHYSICIANS OF LONDON.

WITH LARGE ADDITIONS,

BY
MEREDITH CLYMER, M.D.,
EX-PROFESSOR OF THE INSTITUTES AND PRACTICE OF MEDICINE IN THE UNIVERSITY OF NEW YORK;
FORMERLY PHYSICIAN TO THE PHILADELPHIA HOSPITAL; ETC. ETC.

IN TWO VOLUMES.

WITH A MAP, LITHOGRAPHIC PLATE, AND NUMEROUS ILLUSTRATIONS
ON WOOD.

VOL. I.



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1868

P R E F A C E

TO THE SECOND AMERICAN EDITION.

THE first American edition of this work was out of print in little more than twelve months after publication. So rapid a sale may be accepted as an evidence of its appreciation by the profession of this country, and as a recognition of its claim to being a fair exposition of the Medical Science and Art of the day.

In the present edition the Editor has carefully revised his contributions, and added much new material. His additions are equal to about three hundred pages of the London edition. They will be chiefly found under the heads of: *Lardaceous Degeneration, Vaccination, Measles, Erysipelas, Typhoid, Relapsing, Yellow, and Malarial Fevers, Dysentery, Malignant Cholera, Malignant Pustule, Syphilis, Pathology of the Dietic Diseases, Scurvy, Parasitic Diseases, Rheumatism, Gout, Chronic Bright's Disease, Cancer, Tuberculosis, Diseases of the Nervous System, Diseases of the Heart and Lungs, the Sphygmograph, Pyæmia, Diseases of the Digestive Organs, Diseases of the Kidneys, and Diseases of the Cutaneous System.*

They also include *twenty-two new articles* upon subjects not treated of, or only incidentally mentioned, by the Author, namely:

Camp Measles,
Spinal Symptoms in Typhoid Fever,
Typho-Malarial Fever,
Chronic Malarial Toxæmia,
Chronic Camp Dysentery,
Cholera Morbus,
Cholera Infantum,
Hereditary Syphilis,
Gonorrhæal Rheumatism,
Corpulence,
Physical Diagnosis of the Diseases
of the Brain and Spinal Cord,

Delirium of Inanition,
Chronic Alcoholism,
Epileptiform Neuralgia,
Auscultation in Health and in Disease,
Capillary Bronchitis,
Plastic Bronchitis,
Dilatation of the Bronchia,
Fibroid Degeneration of the Lung,
The Inoculation of Tubercle,
Chronic Pyæmia,
Syphilitic Disease of the Liver.

The subjects of Locomotor Ataxy, Glosso-Pharyngeal Paralysis, Aphasia, Dilatation of the Bronchia, the Sphygmograph and its tracings in disease, were introduced into this text-book by the Editor in the first American Edition (1866). They were first treated of by the Author in the Fifth English Edition (1868), and his articles on these disorders are chiefly condensed from those of the Editor, with the exception of the one on Dilatation of the Bronchia, which Dr. Aitken has abridged from Dr. T. G. Stewart's excellent article in the Edinburgh Medical and Surgical Journal, December, 1867.

The Editor's additions are printed in smaller type, and are thus designated: [].

M. C.

135 LEXINGTON AVENUE,
NEW YORK, September, 1868.

P R E F A C E

TO THE FIFTH LONDON EDITION.

A FAMOUS novel-writer of the day has recorded that “a Preface is a pleasant thing to write, whatever it may be to read.” To indorse the record, and to write a Preface for the *fifth* edition of this Text-book, is very gratifying to the author.

On the 6th of December, 1866 (twelve months after the publication of the *fourth* edition), the publishers wrote to the author, saying, “The state of the stock now renders it necessary for us to request you will prepare a *fifth* edition.”

In accordance with this request, the author has been engaged, during the past fifteen months, in a careful revisal of this work, and the result is that the *fifth* edition has increased in bulk upwards of 100 pages.

This increase implies many more considerable changes (which it is hoped are improvements), as well as additions, than can be made obvious in a preface. They consist, however, mainly, in—

First. The adoption and incorporation in the text of the “New Nomenclature of the Royal College of Physicians of London.” The plan and basis of this nomenclature and classification of diseases is stated at p. 174, vol. i, and a tabular view of the English portion of the Nomenclature of Diseases is substituted, at p. 175, vol. i, for the Nosology of Dr. Farr, which had been used in the previous editions. The reasons for this change are to be found in the account of the “Present State and Aim of Nosology,” at p. 171, vol. i.

Second. The new *English Nomenclature* of the College has been adopted throughout the text. The *definitions* of diseases, and the *foreign equivalents* for their English names, have been incorporated where the several diseases are described.

The author is indebted to the kindness of Dr. Sibson for the privilege of an early copy of the new *Nomenclature* of the College, as it passed through the press, with a view to its being used in this edition.

Third. The subjects of *Malignant Cholera*, of *Paralysis*, of *Epidemic Cerebro-Spinal Meningitis*, and of *Intestinal Obstruction*, have been entirely re-written.

Fourth. The subjects of *Progressive Locomotor Ataxy*, *Progressive Muscular Atrophy*, *Glosso-laryngeal Paralysis*, *Aphasia*, and *Dilatation of the Bronchial Tubes*, the application of the *Sphygmograph*, and its tracings in diseases, where it has been of use, are subjects considered for the first time in this Text-book.*

As with previous editions, so with this one, the author has aimed at giving as fully and faithfully as he could the ideas and the views of the more advanced and able writers of the time, ever desirous that his Text-book should be a "representative book" of the Medical Science and Practice of the day, as actually understood and followed by the best men of our profession.

ROYAL VICTORIA HOSPITAL,
NETLEY, April, 1868.

* [This statement refers to the previous London editions of the work. All of the above subjects were fully treated of in the First American Edition "for the first time in this Text-book" by the Editor.—See *Preface* of the American Editor.]

PREFACE TO THE FIRST EDITION.

IN the compilation of this HANDBOOK I have attempted to give a condensed view of the SCIENCE AND PRACTICE OF MEDICINE. It has also been my object to incorporate and connect the more recently established facts which illustrate the *Nature of Diseases* and their *Treatment* with the time-honored doctrines on which the Science of Medicine has been based.

While the greater portion of the volume is necessarily devoted to a consideration of the *Nature and Treatment* of individual diseases, a more comprehensive range of topics has been embraced, under the title of the SCIENCE OF MEDICINE, than it has hitherto been usual to include in text-books.

The introductory sections indicate the more important element of *General Pathology*; and those principles are shortly stated on which the more modern systems of *Nosology* have been founded since the time of Cullen.

The remainder of the volume, arranged in three divisions, treats, in the FIRST PART, of *Systematic Medicine, Nosology, or the Classification of Diseases*, and suggests that the classification of the Registrar-General of England should be adopted. This statistical nosology, originally proposed by Dr. William Farr, has been carefully discussed and revised at the recent meeting of the Statistical Congress held at Vienna, and a *nomenclature* substantially the same is proposed for adoption in all the States of Europe. The fatal cases are to be *registered* on a uniform plan. A definite *classification*, however, is still undetermined; but I am kindly informed by Dr. Farr that a *classification* nearly the same as the English one has been adopted in Bavaria, and is quietly making its way among practical men in Germany. The Austrians, also, as represented by Dr. Hebra, approve of the separation of *Zymotic* diseases from the others.

IN PART SECOND, under the head of the *Nature of Diseases, Special*

Pathology and Therapeutics, I have attempted to describe the nature of each disease considered as characteristic of its class. In so doing, each disease or morbid process has been defined, not by a logical definition, but merely by stating prominently its leading characters, so that the student may *at once* distinguish the general features of the disease which he has to study, and which the physician has to treat. Having then established the position of each disease in its *Nosological* and *Pathological* relations, those principles are stated which guide its treatment, and in some instances definite details are given.

In PART THIRD, under the head of *Medical Geography*, or the *Geographical Distribution of Health and Disease*, a prominent place has been assigned to a most important department of the Science of Medicine—a subject of study hitherto, so far as I am aware, wholly untaught at our medical schools in this country. It was emphatically written by Cabanis and Malte Brun that climate and natural history lost much of their value from the fact that the physical conditions of the surface of the earth had not then been described in relation to these studies—a deficiency now in a great measure supplied by the labors of Humboldt, Berghaus, and Johnston. So also it may be stated that the NATURE OF DISEASES and their distribution on the globe require that they should be studied in relation to the physical condition of the earth's surface, and to the variation of their *types* in the different regions of the earth. The geographical distribution of Health and Disease in relation to Physical Geography is a branch of the Science of Medicine rapidly and justly growing in importance; and in one department—that of *Sanitary Science*—is beginning to yield most important fruits. To Dr. Mühry in Germany, M. Boudin in France, and Mr. Keith Johnston in this country, the Science of Medicine is largely indebted for the elucidation of this important topic; of which I have attempted to give a sketch, illustrated by Mr. Johnston's map, indicating some of the more useful directions which the study may take.

In my attempt to accomplish this design I have many obligations to acknowledge. In the first instance, the work has its origin in an Article on the "Elementary Principles of Medicine," contributed to the *Encyclopædia Metrópolitana* by the late Dr. Robert Williams, a distinguished physician of St. Thomas's Hospital, London. This Article contains the elements of his classic work on *Morbid Poisons*, completed about sixteen years ago—a work which "occupies the

highest rank in the practical literature of this country ;” and his views regarding their nature are here preserved, commencing from page 185 of this Text-book. It was originally intended by my publishers to reproduce that Article, but I deemed it necessary entirely to re-write and re-model the whole, retaining the statements of facts, and such illustrations as appeared to be of sufficient importance. While I have collected information from every other available source, the limits of this volume prevent me doing more than simply stating at the end of each paragraph the name of the Author from whose writings the statements have been compiled. If I have correctly interpreted and stated the doctrines taught by the veteran laborers and original investigators in the fields of medical experience and research, the names of those of whose writings I have freely and largely availed myself will furnish a sufficient guarantee that the matter I have attempted to communicate is at least orthodox. Much valuable material I have also to acknowledge from anonymous contributors to the pages of the *Medical Journals*. For access to books and libraries I beg especially to express my thanks to Dr. Sieveking, Mr. Martin, Sir James Clark, Dr. Steele, and to the Library Committee of the Royal College of Surgeons. Lastly, my best thanks are due to Dr. Steele, Superintendent of Guy’s Hospital. Notwithstanding the unceasing demands upon his time which the onerous duties of his office entail upon him, he has kindly revised the sheets as they passed through the press ;—for their numerous imperfections I alone must bear the responsibility.

LONDON, 12th October, 1857.

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THE
SCIENCE AND PRACTICE OF MEDICINE.

PART I.
TOPICS RELATIVE TO PATHOLOGY.

CHAPTER I.
OF MEDICINE AS A SCIENCE AND AS AN ART; ITS OBJECTS
AND ITS EXTENT.

THE study of MEDICINE is prosecuted under two relations, namely, as a *Science* and as an *Art*. MEDICINE, considered as a *Science*, takes cognizance of all that relates to our knowledge of diseases; and, especially, of the circumstances under which they become developed, of the conditions of their existence, of their nature and of their causes in the widest sense of these terms. Considered as an *Art* (in so far as Medicine has that practical value), its object is to distinguish, to prevent, and to cure diseases.

The object and aim of MEDICINE as an *Art* is to alleviate human suffering, and to lengthen out human existence, by warding off or by modifying disease "as the greatest of mortal evils," and by restoring health, and even at times reason itself, "as the greatest of mortal blessings." In other words, the practical view required to be taken of MEDICINE is, that "it is the art of understanding the nature of diseases, so as to appreciate their causes, and to prevent their occurrence when possible; to promote their cure, or to relieve them when they occur." (BIGLOW.)

Many branches of human knowledge are combined in the constitution and elucidation of the *Science*; and the practice of Medicine as an *Art* ought to be founded on principles and facts of universal, or at least of extensive applicability.

A consideration of the different topics which together make up the Science of Medicine suggests a division of the subject into the following departments, namely: (1.) PHYSIOLOGY, which embraces the study of the healthy functions of which the human body is the

seat or instrument ; (2.) PATHOLOGY, subdivided into *Special Pathology* and *General Pathology*, which together embrace a consideration of everything relative to the existence and nature of diseases ; (3.) THERAPEUTICS, which expounds the various actions of remedies upon the diseased economy, or the means by which nature may be aided in her return to health ; (4.) HYGIÈNE, which embraces a consideration of the means of preventing disease, or, in other words, of preserving health.

Physiology, General Pathology, Therapeutics, and Hygiène, are sometimes designated indifferently by the titles of the "*Institutes*," the "*Institutions*," or the "*Theory of Medicine*."

These departments of science are all preliminary subjects of study, and constitute a necessary and appropriate introduction to the Practice of Physic, in which Special Pathology and the treatment of special diseases are the leading topics of consideration.

Each of these departments has grown or expanded itself into a great branch of science ; and any single section is sufficient of itself to occupy the lifetime of an individual in working out and studying it in detail. It is therefore not possible for the human mind to embrace all of these departments in their whole extent or relations to each other ; and, setting aside the consideration of theories and systems, it has been truly observed, "that no man possesses all the pathological knowledge contained in the records of his art" (CHOMEL) ; and it is, therefore, far less possible to embrace in any single treatise a view of the Science of Medicine in all of these departments.

For the purpose of teaching the Science of Medicine in its application to practice, its elementary principles, as developed in the departments of Pathology, are the most useful guides to the student.

CHAPTER II.

HOW THE PROVINCE OF PATHOLOGY IS MAPPED OUT.

AN inquiry into the nature of diseases embraces a consideration of the following topics : (1.) *The accurate observation and correct registration of facts in Pathology.* On the efficiency of the machinery devised for these important ends will rest our power, to curb the invasion of our science by the guesswork of theory. Such records must be the means eventually of rooting out the traditional errors which so largely pervade medical literature. (2.) *Descriptive Pathology, embracing GENERAL and SPECIAL Pathology.* Special Pathology is intended to comprehend a consideration of the essential nature and origin of particular diseases as they occur in man and animals, and General Pathology to include those more general facts or principles which result from a comparison of particular diseases with each other. Although Special Pathology comes first in the order

of Nature, yet, wherever the arrangements for Medical education are complete, General Pathology is taught as an introduction to, or conjointly with, the special study of diseases, just as in other sciences—for example, in chemistry—it is found convenient to give a general view of the principles which have been established by experiment and observation, before entering upon the particular details of the science. All theory in Medicine; all descriptive Pathology; all grounds for rational speculation regarding the nature of diseases, and for the framing of experiments; as well as all maxims of practice which aim at the prevention or cure of diseases, must rest ultimately on observed and recorded facts. *Accuracy of observation* is therefore the first lesson the student has to learn in all methods of investigation, and the lesson is one of paramount importance. The best observations, however, will avail but little unless the observed facts are recorded in such a way as to secure their preservation; and descriptive pathology mainly concerns itself, in the first instance, with the *Registration of facts*, as embraced,—

- (1.) In the *History of cases of disease* from their origin to their end.
- (2.) In the *Statistics of disease*. Such registration includes methods for preserving, in an authentic and permanent form, the memory of facts in Pathology as they occur. It thus eventually furnishes materials upon which future Pathologists and Statists will build a comprehensive and definite system of scientific Medicine. It will furnish the means of teaching all that is necessarily involved in our notions regarding the nature of diseases. The descriptive Pathology so arrived at considers diseases as they exist, or have existed in man, in the lower animals, or in plants. It considers the conditions under which diseases originate; it considers how far certain conditions are fulfilled before disease establishes itself; and it aims at demonstrating how far such conditions are inconsistent or incompatible with the maintenance of health. Subsequently, with extended information, descriptive Pathology may undertake to assign the conditions which give rise to certain diseases rather than to others. It will eventually define the elements necessary to establish, to originate, or to constitute particular diseases; and will show how the same disease, or class of diseases, may assume various forms, but in all of which definite elements are recognizable. Descriptive Pathology thus aims at determining and describing the essential elements of a disease.
- (3.) *Speculative Pathology* assumes that we know what a disease is—that we know the effects it produces—that we know the conditions necessary for its existence—that we know its relations to other diseases. It seeks to inquire how certain conditions or circumstances will operate in bringing about disease. It seeks to determine the tendency in the future of a diseased state from certain observed facts in its course, or in the course of similar diseases. Statistical data are thus the main basis of its operation.
- (4.) *Pathology dictates the maxims of rational practice*. It is in the nature of the science of Pathology that it always ought to be in advance of our certain knowledge regarding the treatment of diseases. It is the basis of rational medicine; for it is rational to know the nature of a disease, in order

(1.) To enable us to prevent

it; (2.) To understand the principles which ought to guide us in the management of it. Such are the main divisions which the province of Pathology embraces.

It is intended, however, in the first part of this handbook merely to guide the student to notice,—(1.) The *relative* nature of the terms “*Life*,” “*Health*,” “*Disease*,” (2.) How the nature of diseases may be elucidated; (3.) The nature of the morbid phenomena, symptoms, and signs of disease; (4.) The means and instruments of investigation into the nature and causes of disease; (5.) Some of the more elementary constituents of disease; (6.) Some complex morbid states associated with individual diseases, or with conditions of ill-health (*cachexiæ*); (7.) The modes by which diseases terminate fatally; the types of disease and their tendency to change; (8.) The general treatment of the complex morbid processes.

In the three subsequent parts of this work it is intended to consider some of the details of the science and practice of Medicine, to furnish the student with,—(1.) A nosological system by which to classify and name diseases. (2.) A detailed description of characteristic diseases in the respective classes of that nosological arrangement. In this part a definition (provisional) and a history of the nature of each disease will be given; the probable course and succession of events will be described, and the grounds on which an accurate diagnosis may be made, or a prognosis expected; and, lastly, a detailed account of those rational modes of treatment which are consistent with the established principles of the *Institutes of Medicine*. (3.) An account of what is known relative to the geographical distribution of diseases.

CHAPTER III.

RELATIVE NATURE OF THE TERMS LIFE, HEALTH, DISEASE.

THE word *Disease* is used in a general and also in a specific sense; as when it is said that a person is diseased, without the nature of the affection being stated; or, that he suffers from a particular disease, such as small-pox. Attempts to give a precise definition of the term *Disease* have all been unsuccessful. The relations of the morbid state to the condition of health, and of health to the performance of the vital functions, are of such a kind that they can merely be described in connection with each other, but not defined.

If *life* is understood to imply an active state, resulting from the concurrent exercise of the functions of the body, then there are conditions of activity and of mutual adaptability of functions and of parts, both as regards body and mind, which are necessary to healthy existence. Our notions of *the conditions of health* have thus considerable latitude. *Health* is merely a name we give to that

state or condition in which a person exists fully able, without suffering, to perform all the duties of life. Many degrees of this state are therefore at first sight obvious, from the possession of a feeble existence to the most robust condition of the body; and there are many degrees of feebleness and delicacy of health which we cannot say are due either to disordered or diseased states of the frame. Our notions of *normal life* are thus so extremely indefinite that it is only by a forced abstraction the normal can be separated from the abnormal. Hence also our idea of *disease* is very indefinite; it cannot be separated by any well-defined boundary from our idea of *normal life*, and the two conditions are connected by a kind of debatable border land.

When we regard, therefore, the phenomena of the living state and the conditions of health, we can readily observe when and how *disease* is but a deviation from the state of health, consisting for the most part in a change in the properties or structure of any tissue or organ, which renders such tissue or organ unfit for the performance of its actions or functions according to the laws of the healthy frame.

It is now a received pathological doctrine that *disease* does not consist in any single state or special existence, but is the natural expression of a combination of phenomena, arising out of impaired function or altered structure. All attempts, therefore, to define *disease* by the use of such terms as "*derangement*," "*modification*," "*alteration*," "*change*" from the pre-existent state of health, show, in the first instance, that very various ideas are attached to the term or to the state; and, secondly, that these terms point to a nosological division into structural and functional disease, rather than to a state common to all forms of disease.

A definition of any state of disease ought to include all the circumstances, whether functional or organic, which constitute the deviation from health; and for very obvious reasons such a definition can only be approximately expressed.

CHAPTER IV.

HOW THE NATURE AND CAUSES OF DISEASES MAY BE ELUCIDATED.

THE nature of the derangements to which the human body is liable may be studied under the three following aspects: (1.) As diseases present themselves in individual cases, becoming thereby the subjects of CLINICAL INVESTIGATION and INSTRUCTION—a method of teaching in which the *Natural History* of the disease ought to be a special subject of study; (2.) As they constitute particular genera or species of disease, forming the topics of SPECIAL PATHOLOGY; (3.) As they may be reduced to and studied in their primary elements, forming thereby the science of GENERAL PATHOLOGY.

But, in whatever aspect we may view disease, there is invariably presented to the student the same subjects for investigation, namely, —*First*, The morbid phenomena or symptoms by which we become aware that derangements have taken place in the economy. It is by a mental effort that either the student or the physician converts these symptoms into signs of disease; and hence arises the necessity of studying *Symptomatology* or *Semeiology*. *Second*, The agents by which derangements and diseases are produced, generated, or brought about, constituting the department of *Etiology*. *Third*, The seats or localities of disease, or of derangements, constituting *Pathogeny*. Here the peculiar nature, general forms, and types of disease must be studied, together with varieties in their course, duration, and termination. *Fourth*, The morbid alterations discoverable in the structure of the body before, but more especially after death, constituting *Morbid Anatomy*. These alterations must be studied in connection with the symptoms, the causes, and the course of the disease. *Lastly*, The elementary constituents of diseased products, constituting *Morbid Histology*, must be recognized in the first instance, and contrasted with analogous constituents of the body in the healthy state.

CHAPTER V.

OF MORBID PHENOMENA, SYMPTOMS, AND SIGNS OF DISEASE.

It has been stated that only by a mental effort is the student or physician able to convert *symptoms* into *signs* of disease. Therefore the idea associated with "*sign*" is of a much more comprehensive kind than that which is connected with the word "*symptom*:" the former implies the possession of more extensive knowledge—a knowledge such that comparisons may be instituted amongst the symptoms which present themselves. Certain symptoms of disease, or of disordered function, are thus recognized to be peculiar, characteristic, or *significant* of a particular morbid state. A symptom is thus converted into a sign, and what is called a *diagnosis* of the disease is made.

Symptoms and signs of disease derive their importance from the fact that they are capable of being connected with lesions of structure or disorders of function; and both of these conditions mutually act and react upon each other, and thus they greatly aggravate the expression of general disease. In place of the concurrent exercise of function, and the mutual co-operation of parts in a state of health, both as regards mind and body, we have *symptoms* of disease expressed in various ways, characteristic of the function at fault, and incompatible with the normal existence of the part or organ affected, or of the body generally. From such phenomena the physician makes up his mind,—(1.) As to whether or not disease

exists. (2.) How far the condition of the patient is removed from the state of health usual to him. (3.) As to the nature of the disease, and how it is distinguished from other ailments, or in what respects it may differ from the same ailment in other people in similar circumstances. Thus a *diagnosis* is made by the art of converting *symptoms* into *signs* of disease.

But the physician at the same time generally carries his mental exertion a little further. He tries to arrive at a just estimate of the probable result or *event* of the malady, and so makes up his mind,—(4.) As to whether the illness will terminate in the death of the patient, in permanent organic mischief of greater or less extent, in persistent impairment of the general health (*cachexiæ*), or in complete recovery. As in Politics, so in the science of Medicine: the Politician and the Physician have each to deal with the future, as well as with the present. Both endeavor to *forecast* events; and thus, in the practice of Medicine, we are said to make or give a *prognosis*. (5.) The Physician must be able also to appreciate with reasonable rapidity those *symptoms* which are peculiar, and to recognize them when associated together as the *signs* of particular or definite morbid states. Such *symptoms* are then said to furnish *pathognomonic signs* of disease. (6.) The Physician must further discriminate, and try to put a fair and just value or interpretation upon, those symptoms which are only experienced by the sensations (subjective) of the patient himself, as contrasted with those which may be seen or appreciated by others—such as objective phenomena or physical signs.

The interpretation of symptoms can only be successful after a close observation of the patient—often prolonged, and repeated for more complete investigation—so as to connect the results arrived at with his previous history. The utmost logical acumen is required for the due interpretation of symptoms. The individual value of each ought to be duly weighed; one symptom must be compared with another, and each with all; while the liability to variation of a similar symptom in different cases of a like kind must not be forgotten; and the occasional absence of the usual pathognomonic signs may be sometimes calculated upon. Thus only can the nature of a disease be clearly determined—its severity and dangers fully appreciated—its treatment indicated, and the probability of recovery foretold.

A close observation of general symptoms, in all their details, is absolutely necessary; and the investigation is aided practically by the improved instruments of the present day, and better methods of examination. Above all things, *methodical examination* is essential for the student, if he would acquire the habit of carefully and accurately examining the nature of the cases of disease with which he will have to deal. Patients must be examined methodically, in order that the symptoms of disease may be correctly interpreted, and that nothing be overlooked or neglected. Directions have been given by many authorities for acquiring and habitually following a definite system of examining patients, as to what are the essential data to be obtained and recorded in case-taking; and

although, as Dr. Acland justly remarks, a skilful practitioner can learn the truth of most cases in any order, or in no order, yet it is highly desirable that a regular order should be followed by learners; and all cases observed by the student should be methodically entered in a note-book for the purpose. This habit will thus eventually become a necessity, and will be found most useful in after-life, and especially in consulting practice.

The following works are recommended for study, and as guides for acquiring the best methods of observing and recording cases: (1.) *A Manual of Medical Diagnosis*, by A. W. Barclay, M.D.; (2.) *A Handbook of Hospital Practice; or, an Introduction to the Practical Study of Medicine at the Bedside*, by Robert D. Lyons, M.B., Professor of Medicine in the Catholic University of Ireland; (3.) *An Introduction to Clinical Medicine*, by John Hughes Bennett, M.D., senior Professor of Clinical Medicine in the University of Edinburgh; (4.) "Suggestions for Taking Cases," by Dr. Beale, *Archives of Medicine*, vol. iii, p. 47.

CHAPTER VI.

MORBID ANATOMY AND PATHOLOGICAL HISTOLOGY: THE SPECIAL MEANS AND INSTRUMENTS BY WHICH THE NATURE OF DISEASES MAY BE INVESTIGATED.

MORBID, or, as it is also sometimes called, PATHOLOGICAL ANATOMY, is that department of medical science which treats of the changes produced by disease in the solids and fluids of the body; while MORBID or PATHOLOGICAL HISTOLOGY treats of the origin, development, growth, and decay of the new products or new formations which are the elementary constituents of structural or organic lesions. The anatomy of diseased parts stands in the same relation to the development of morbid phenomena and conditions of disease that the anatomy of healthy structures and the histology of the textures do to the natural functions and process of development, growth, and nutrition in the healthy body.

The vestiges left by the prolonged existence of a morbid state, whether in the body of man or of the lower animals, have always claimed from the physician a large share of attention; and in proportion as the knowledge of healthy anatomy and physiology has become extended and prosecuted in all its bearings, so has pathological science been extended, and morbid anatomy has gradually but steadily acquired an important and prominent position among those branches of study on which Medicine rests its claims as a science.

MORBID ANATOMY is a department of medical science which has gradually grown out of the accumulated experience and observation of ages; but PATHOLOGICAL HISTOLOGY, as a science, is of modern

origin. It is but yet in process of development, although its foundations may be traced in the works of the earliest medical writers of antiquity. All of them refer to changes which they *merely supposed* had taken place in the internal organs; and they were doubtless led to this assumption by observing the connection that existed between structural lesions of the external parts and their accompanying symptoms. Hippocrates describes the deposit of tubercles in the lungs, the symptoms occasioned by them in a crude state, and those which attend their softening and discharge.

The science of MORBID ANATOMY is a record of facts. In its relation to the progress of Medicine it is a living record—a history whose pages must be ever open to receive the observations which are constantly being made by those engaged in pathological pursuits—a record from which one may ascertain at any time the conditions under which morbid changes or new formations in the body have taken place. The pages of this history show that at the present day the department of pathology is in a transition state; and the position of Medicine, as a science, must eventually result from a re-arrangement of the innumerable details which the sciences of morbid anatomy and histology may disclose and unfold. It is necessary, therefore, and often advantageous, to look back upon the past, and see what has already been done, so that its venerable facts may not be lost sight of, but grouped in series with the extensively verified experiments and observations of the present day. In so doing, if we pause and contemplate the steps which have been taken to arrive at our present position, such a contemplation may stimulate the youthful student to the noblest exertions of his intellect, as he can not fail, with extensive study, to see before him, and on every side, much unlabored but productive soil. Such a retrospect will at the same time have the effect of placing in a prominent aspect the varied influences which morbid anatomy has had on the science of Medicine, the conditions under which it has flourished, and the legitimate objects of its investigations.

The art of printing had not been long invented when books on morbid anatomy began to issue from the press; and although, the early period of the fifteenth century has left little enduring literature of any kind (but has been mainly distinguished by the number of colleges then founded), yet about this time pathological anatomy in the medical school of Florence shows the earliest evidences of an existence.

The facilities for study which the art of printing introduced soon stirred up ardent students; and the sixteenth and seventeenth centuries produced much that will ever remain famous in the annals of medical science. Eustachius, Tulpius, Ruysch, Harvey, Malpighii, and Leuwenhoeck are names familiar as household words to the student of medicine. The earlier attempts of this period to form a system of pathological anatomy is characterized by abortive endeavors to explain all results upon some exclusive and general principle. A spirit of speculation marks the character of the age. The men of that time had observed but few facts; and on these facts they preferred to speculate and dogmatize, rather than prosecute the

further interpretation of nature, or record more observations. Accordingly, theories in abundance successively led captive the minds of the medical world, and, disappearing one after the other, demonstrated the unstable nature on which the science of Medicine had been placed. The leader of each sect founded his so-called school or system, all of them distinguished by a due amount of arrogance and contempt for predecessors and contemporaries—a feeling unhappily not yet quite extinct. The “*vital agency*,” the “*influence of the humors*,” and of the “*solid organs*,” have each been considered by turns as the only orthodox belief; and each has had their school and sect, respectively designated as the *Vitalists*, the *Humoralists*, and the *Solidists*. The theories of Galen, of Paracelsus, and others, have all been famous in their time, but are now unheard of, and almost unknown. The same fate awaits the false theories and absurd conceits of more recent origin, although, as in the case of Stahl, Cullen, Brown, and Broussais, they have had a wide prevalence in the schools of Europe, and made impressions on the sentiments of the profession which yet influence their modes of practice and the reasons of their belief. Broussaisism, Hahnemannism, and some other systems, “the fruits of a luxuriant fancy and of few facts,” must all descend, as others have done, the same inevitable slope to oblivion; but the vast collection of facts which the founders and followers of such systems eventually accumulate and bring to notice, remain unchangeable, and will continue to recur in the daily experience of our profession, just as they appeared to the venerable fathers of medicine centuries before the Christian era. The practice of medicine, as based upon rational principles and a knowledge of the nature of diseases, has oscillated through all these systems and theories, and the science of morbid anatomy has been marked throughout by unmistakable periods of *progress*, of *stationary existence*, or even of *retrogression*, according as one or other exclusive system had the ascendancy, or as each principle of practice challenged for itself a supreme importance.

The modern doctrines relative to the nature of diseases and the practice of Medicine may be said to be guided by the dictates of *Physiology*, and what is known regarding the development of the human body. Ordinary dissections alone, or post-mortem examinations of the body, have long since ceased to furnish us with facts before unknown; and new modes of extending observation and research, by taking advantage of every physical aid to the senses, are diligently looked for by the modern anatomist, physiologist, and physician; and the means and instruments which advance the science of physiology are well able to advance our knowledge regarding the nature of disease-processes.

A belief is now rapidly gaining ground, and acquiring a hold on the popular mind, that advances in the science of medicine in future years will be mainly due to a *better appreciation of the causes of disease*; and just in proportion as our knowledge of physiology and pathology becomes more exact and extended, so will the *causes of disease* be appreciated, and the *occurrence of disease* on a large scale prevented. An amiable and large-minded physician, Sir John

Forbes, who but recently has taken his place amongst the "Great ones of the Past," emphatically recorded the observation more than fifteen years ago, that "here the surest and most glorious triumphs of medical science are achieving, and are to be achieved." He himself lived to see great and good results; to see improvements in social and sanitary matters which continue to be realized, and whose rapid progress is characteristic of the present period. Within the last half-century land-draining and town-sewering have ripened into sciences. From rude beginnings, insignificant in extent, and often injurious in the first instance, the systematic sewerage of towns and draining of land have become of the first importance. Land has thus, in not a few instances, doubled its value. Town-sewering, with other social regulations, have contributed to prolong human life from 5 to 50 per cent. as compared with previous rates in the same district. Agues and typhoid fevers are reduced in the frequency of their occurrence. Since 1840 an annual mortality in English towns of 44 in 1000 has been reduced to 27; an annual mortality of 30 has been reduced to 20, and even as low as 15. Not less remarkable reductions have taken place in the mortality and loss of strength in the army and navy; so that generally it may be said that human life has now more value in England than in any other country in the world—a result entirely due to better sanitary arrangements (Rawlinson "On Sewering of Towns," *Soc. of Art Journal*, vol. x, p. 276).

The political economist can not now, therefore, regard MEDICINE in any other light than as a productive art; and the labors of the Physician, whether in civil or in military life, can not be regarded as unproductive labor.

But the science of Physiology (on which much of our sanitary improvements are based) has immeasurably outstripped the science of Pathology in the comprehensiveness of its views and in the value of its results; while Pathology, in its turn again, has always been, and ought to be, in advance of Therapeutics. The best physiologists have distinctly recognized that the basis of the science must include not only a knowledge of animals below man, but a knowledge of the entire vegetable kingdom. Without such an extensive survey of the whole realm of organic nature, we cannot possibly understand human physiology, and far less comparative physiology. The science of Pathology, therefore (whose aim is to expound the *nature of diseases*), must be, *à fortiori*, very far behind. The diseases of the lower animals, for instance, rarely form any part of the study of the student of Medicine. The diseases of plants are almost entirely neglected. Yet it is clear that until all these have been studied, and some steps taken to generalize these results, every conclusion in pathology regarding the nature of diseases must be the result of a limited experience from a limited field of observation. How do we know that the blights of plants, or the causes of them, are not communicable to animals and to man? We know how intimately related the diseases of man and animals are with famines and unwholesome food; and of famines with the diseases of vege-

table and animal life, as much as with the destruction and loss of food. (See chapters on Zymotic diseases.)

To physiology, therefore, in its most comprehensive sense, and to a knowledge of the natural and normal development of animal and vegetable beings, we must look for future progress in pathology; while the means and the instruments which advance physiology will simultaneously advance our knowledge regarding the *nature of diseases*,—a sound knowledge of which can alone enable us to “*appreciate their causes*,” and so arrange measures for the *prevention of many of them*, based on the great truths of science.

Organic chemistry, the microscope, the ophthalmoscope, and such like instruments, have opened up new fields of labor, which are being diligently cultivated; and while alterations in the ultimate tissues and organs are more especially attended to, the first beginnings of disease, the development of new formations, and the examination of excretions, claim a large share of attention.

Histology, or the study of the development and arrangement of the tissues in the formation of normal and healthy organs, is characteristic of the anatomical investigations of the present day; while the histology of morbid products and chemico-physiological investigation into the nature of morbid changes is characteristic of the pursuits of the science of modern PATHOLOGICAL ANATOMY.

It is also a significant fact that now, in the nineteenth century, some of the leading doctrines of the *humoral* pathology which prevailed in the seventeenth, are again revived. The experience and learning of that erudite period are now being made available for modern uses. By the improved means, instruments, and methods of research of modern times, important truths may be sifted from the errors and theories with which they are mixed up in the ancient chronicles of medical science; and when we get analogous conditions of disease with which the phenomena described by the ancients may be compared, “not a few of the apparently modern beliefs are daily found to have a time-honored reputation unappreciated before.”

The chemist and the histologist now combine their researches, and work hand in hand; and we regard them as the most inquisitive anatomists of the time. They lend assistance of the most important kind in working out the foundation of our knowledge regarding the nature of diseases, the details of which can only be made more certain and perfect by taking advantage of every kind of scientific knowledge which can be brought to bear upon medical research, and more especially,—(1.) By physical aids to the senses, extending our means for the actual inspection and appreciation of phenomena. The use of the stethoscope, of the microscope, thermometer, ophthalmoscope, laryngoscope, and specula of various kinds, aided by a careful study of the writings and labors of the men who have more particularly devoted their attention to observations by such means, may be quoted as examples (LAENNEC, LOUIS, WALSH, STOKES, HOPE, BENNETT, QUEKETT, VIRCHOW, WUNDERLICH, TRAUBE, VOGEL, BEALE, GRAEFE, CZERMAK, and others). (2.) By the knowledge (gradually being made more extensive) of

the textures, organs, and functions of the body whose normal exercise constitutes a healthy existence (LONGET, MULLER, SHARPEY, VALENTIN, ALLEN THOMSON, CARPENTER, KIRKES, PAGET, KÖLLIKER). (3.) By an intimate knowledge of the normal development of the human textures, as well as those of plants and animals from the fecundated ovum (BISCHOFF, COSTA, ALLEN THOMSON, HUXLEY, NEWPORT, and KÖLLIKER). (4.) Besides these kinds of investigations, the science of practical medicine has been, and is being, advanced by operations and experiments upon the internal organs of living animals, opprobriously termed *vivisections*. At some of our great schools of medicine such investigations are now being actively but judiciously prosecuted and taught; as by Bernard, in Paris, Drs. G. Harley, Brown-Séquard, and Pavy, in London.

Successful inquiries into Pathology, or the nature of diseases, cannot be said to have commenced till the middle of the eighteenth century, when the great work of Morgagni issued from the press. It was the work of his lifetime. In the eightieth year of his age, and not till then, did he consider himself warranted to publish his observations, *De Sedibus et Causis Morborum* (1761); a work whose material and circumstances of publication read us the practical lesson, that the more frequently a disease occurs, the more necessary it is that its phenomena should be carefully investigated. And when we think of the prudent reserve, the anxious and the conscientious delay exhibited by Harvey, Morgagni, and Jenner, in the publication of their respective researches, we cannot but contrast the circumstances with those under which the exuberance of medical publications are now given to the world. Morgagni modified and corrected many of the views entertained and promulgated by his predecessors; and the study of the nature of diseases was carried into the commencement of the present century by CULLEN, DE HAEN, WILLIAM and JOHN HUNTER, PORTAL, and BICHAT.

The knowledge of the physician regarding the nature of disease-processes may now be observed to have advanced simultaneously with that of *general anatomy*; and when the component parts of an organ, and of the human body, came to be distinguished, it was soon observed that membranes and tissues might be individually diseased while neighboring membranes and tissues remained untouched. Bichat's idea, therefore, of decomposing the animal body into its elementary parts, must be regarded as the foundation of modern special pathology; and while he pointed out the necessity of studying diseases with reference to the different tissues as separately and specially affected, it has been since shown, in a remarkable manner, how general anatomy, deduced from physical properties of parts and crude observation, may coincide with more minute investigations of a chemical and microscopical kind. The membranes and tissues composing the organs of the body, roughly torn asunder by Bichat, are now themselves being daily subjected to a more inquisitive analysis of an anatomical and chemical nature, which unravels them into still more minute histological elements.

Although, therefore, Bichat entertained the view that each tissue had its own *diathesis*, it is to Cullen and the Hunters in this country

more especially that the application of the distinction of tissues was made to illustrate the nature of disease-processes.

Cullen's descriptions of diseases are descriptions of groups of phenomena which comprise complex morbid states.

The written labors of the Hunters form but a small part of the memorials of what they did to elucidate the nature of diseases, and it is only those who have had the opportunity of carefully examining their museums, preserved in London and in Glasgow, that are able to form any conception of the comprehensive nature of their labors, or to assign to them a proper place among those who have successfully advanced the science of Medicine. They hold a position at least one hundred years in advance of the age in which they lived. Bichat, Cullen, and the Hunters, in their respective countries, have thus reciprocally influenced and advanced the progress of our knowledge regarding the nature of diseases. And although it was reserved for Bichat to complete a more perfect system of general anatomy, it must not be forgotten that Dr. Carmichael Smith, in 1790, applied his knowledge of textural anatomy to elucidate the nature of disease-processes; and that Pinel, after him, in his *Nosographie Philosophique*, made the distinction between the membranous and other animal structures the foundation of his pathology. The classic work of Baillie (his *Morbid Anatomy*), published in 1793, closed the labors of the past century.

If now we look to the tendency of the studies and researches of those men just named, including Bichat, we shall find the truth gradually being more fully appreciated, that it was necessary to study alterations of structure so as to connect morbid changes with the symptoms of diseases during life, and with the operations of ascertained causes of morbid action. The nature of the morbid changes was now observed to be more apparent in the progress of external diseases; and therefore surgical experience was brought to bear upon the elucidation of internal disease-processes.

Thus the progress of Morbid Anatomy is, in a great measure, a record of the history of Medicine; and we can trace the science of special morbid anatomy, giving a character to the various systems of the healing art which have prevailed from time to time.

All the writers up to the time of Bichat, Laennec, and Abercrombie, were pure morbid anatomists, who did not connect the effects of disease with their causes, and who recognized the changes of disease as important only in proportion to their magnitude as apparent to the senses. They are therefore regarded as pure solidists, whose researches doubtless contributed much towards a correct knowledge of the changes in the organs of the body, while the condition of the fluids was neglected, as well as the relations of the texture, organs, and fluids, in the combined exercise of their functions. Simple functional disturbances were thus wholly overlooked, and the constitutional connection of local affections entirely lost sight of.

The contemporaneous surgery of the period previous to Bichat was marked by its unwillingness to recognize anything but material facts, mechanical processes and contrivances. The surgeons of those

days desired to know nothing but anatomy and mechanics; and, accordingly, it may be recognized as the period of pure anatomical and mechanical surgery, distinguished by the writings of men whose works bear ample testimony that the surgery of the period was founded on exact and even minute anatomical knowledge. No allusion is made, however, by them to medicine,—they make no application of physiological truths, and they encourage no therapeutic tendency apart from mechanical or instrumental interference.

The purely solidist, as well as the purely humoral principles by which the nature of diseases have been explained, may be said to have died a natural death long ago; but, as already noticed, the remembrance of what is valuable in the results of both are preserved in modern pathology, which takes its stand upon anatomical and physiological facts, connected by simple methods of inductive observation with the symptoms and signs of disease as seen and expounded to the student by the distinguished professors of Clinical Medicine at most of our celebrated schools, where Clinical Medicine is taught.

In this field of instruction it seems invidious to mention here the names of men still living. For their own sakes, as well as for science, may they be long deprived of being thus honorably and respectfully mentioned. As teachers, they are in our own country familiar to every student. As recorders of what they observe at the bedside and after death, they are not less celebrated abroad than appreciated at home.

Tested by extensive clinical observations, the character of the present period in the history of Practical Medicine is one of *probation* as well as of *progress*, marked by a close inductive examination of past generalization and classification of facts, however remotely connected, which illustrate the nature of diseases and their treatment.

Side by side, since 1816 and 1819, the microscope and the stethoscope, under the influence of such men, have advanced our knowledge of the nature of diseases with a regular and accelerated velocity; but they have only done so as assistants and in subordination to laws and facts whose knowledge we have acquired by a close observation of general symptoms, of which such instruments have never been intended to take precedence. They have never accomplished, nor can they ever accomplish, useful practical results, to the exclusion of such other methods of observation as have just been noticed. We are not to confound *relative* smallness with *absolute* simplicity, and believe that because a simple organic cell is a small object—because we can see around it, through it, and on every side of it—the functions and conditions of its existence are less *complex* or less obscure on that account than are those of a more complex organ, or the functions of a living body.

We are not to suppose that because the stethoscope enables us to detect a mitral murmur, or a crepitation in a lung, we are justified at once in adopting one, and only one, method of treatment. It is this exclusive use of instruments, to the disregard of general

symptoms and signs of disease, derived from close observation and knowledge of the living functions, which leads to the repudiation of the use of such instruments by the sagacious and experienced physician, who sees the numerous errors not unfrequently committed by his younger brethren, who trust too exclusively to these instruments in the diagnosis of disease.

Like the stethoscope, the microscope has been unjustly and unnecessarily burdened with labor, and has been equally unjustly blamed, and brought into unmerited discredit, when it has failed to elucidate the nature or even presence of a morbid state, the existence of which could not be doubted, but which the sense of sight could not appreciate, even when presented in small quantities greatly magnified. In such instances the microscope has been applied to uses which it is not the nature or province of the instrument to detect. The gravimeter or hydrostatic balance, the microscope, the stethoscope, the ophthalmoscope, the laryngoscope, the pleximeter, and the thermometer, are merely instruments of pathological inquiry, each one adapted for the determination of particular classes of facts. They can only elucidate disease when they are brought to bear upon physical properties, the nature of which they are able to appreciate; and it is only from their *combined and appropriate* use, in connection with a history of the general signs and symptoms in each particular case, that our knowledge of the nature of disease will be advanced.

The industrious employment of these aids to diagnosis, and an intimate acquaintance with the results, are attended with this further advantage, that such practice and knowledge enable their possessor to appreciate the general symptoms of disease with infinitely greater certainty than heretofore. This is the usual consequence of training in all exact methods of observation. The thorough study of these aids to the senses in appreciating disease leads directly to the possibility of dispensing with them in many instances. By means of auscultation and percussion, for example, our attention has been drawn to numerous conditions of the thorax, which enable us to make the diagnosis at the first glance, which hitherto was not possible; because the conditions for diagnosis could never have been recognized without such physical aid to the senses as that derived from auscultation and percussion. In many cases, from the mere inspection of a patient, a well-instructed clinical student may decide upon the existence of *pleurisy*, *pneumothorax*, *emphysema*, or *pulmonary tubercle*. The initiated are thus frequently enabled to dispense with percussion and auscultation; but if they had never acquired the practical knowledge of the subject—if they had never examined numerous patients by means of these physical aids to diagnosis—and so learned thus to determine with great exactness the significance of the various forms and movements exhibited by the thorax, they would never have been able to appreciate their significance. So, also, the physician well instructed in the use of the thermometer may, in hundreds of cases, without its aid, draw conclusions with great certainty, incomprehensible to others not so instructed; but if, led away by this skilfulness, he is induced to dispense with *exact*

thermometrical control, he may soon fall into gross errors. So it is with the ophthalmoscope, specula, and all other more or less exact physical aids to diagnosis. Let them be in constant and appropriate use, but the results must always be taken and compared in connection with other general symptoms of disease.

In all the temperate regions of the world, histology, as applied to morbid products, has been cultivated, and has advanced our knowledge regarding disease ever since 1838. In warmer latitudes our knowledge of practical Medicine has been advanced by extensive observations on physical climate, medical topography, and by organic chemical analysis applied to obtain therapeutic agents from the vegetable world. Those may be said to be the characteristics of the researches of our own country, Germany, France, and America, as contrasted with the nature of the observations mostly prosecuted in India.

No exclusive doctrine will now stand the test of well-directed pathological inquiry, the main object of which is to connect all organic changes (lesions) and functional derangements with their symptoms and causes, with the view of applying rational remedies and prophylactics. The too exclusive study of pure organic pathology and morbid anatomy leads to no distinction between the signs and causes of disease; and the obvious tendency of such exclusive study is to exaggerate the importance of the principles it may establish, to hold out no hopes of cure, and to undervalue the power of remedies and remedial measures. To obviate this tendency it is necessary to have recourse to inductive reasoning, so as to connect all the morbid changes seen or appreciated after death with the signs and symptoms of disease observed during life. Thus it is that links in the chain of disease-processes which, from a one-sided or exclusive view, appear isolated and localized, are really found to be connected with each other. It may be, also, that they are connected with a long but intelligible series of processes developed during life through the metamorphosis of tissue, and going on in apparent health, or in an obviously morbid exercise of function. The constitutional origin of many local diseases, otherwise inexplicable, then becomes apparent.

Among the more eminent exponents of this rational school of pathology, who at an early period in this country discerned and appreciated such doctrines, we find the names of ALLEN, GOLDING BIRD, SIR ROBERT CARSWELL, GREGORY, HOPE, HODGKIN, MARSHALL HALL, PROUT, WILLIAM STARK, JOHN THOMSON, TWEEDY TODD, and many others, who, although now no more, have left behind them imperishable evidence of their labors. The younger pathologists of the present day, whose name is *Legion*, follow in the footsteps of these men, extending the fields of observation and the boundaries of the science of Medicine. By them the importance of morbid anatomy is sufficiently appreciated, and its province distinctly defined and limited as follows, namely: (1.) To detect the changes which have taken place during the course of diseases in the structure of tissues and organs of the body; (2.) To demonstrate the

exact seat of local alterations established during the progress of disease.

The investigation and elucidation of the *nature, course, and causes* of those changes, constitute the prominent objects of the science of pathology. By the aid of morbid anatomy and clinical observation during life, pathology seeks to establish the relations of the changes which lead to the lesions, and so to connect the general progress of disease with its symptoms and signs.

MORBID ANATOMY goes beyond its province when it attempts to point out the nature of the proximate cause of disease. It is only by the application of inductive reasoning that the connections of causes and morbid effects can be shown, and such constitutes the main object, and is the highest aim of the science of PATHOLOGY.

The *morbid anatomist* finds a lesion or change for what ought to be the natural structure, appearance, or condition of a part. The *pathologist* seeks to connect such lesions with signs and symptoms during life, that the *practical physician* may suggest a remedy to the disease, and that the *nosologist* may give it a name, distinguishing characters, and a place in his classification.

CHAPTER VII.

THE ELEMENTARY CONSTITUENTS OF LESIONS AS SHOWN BY MORBID ANATOMY AND OTHER MEANS OF RESEARCH.

WHERE the material effects of disease can be rendered obvious they are found to consist for the most part of,—

1. Morphological changes in the elementary textures of the body generally, and altered conditions of the fluids.
2. The presence of new formations foreign to the normal condition of an organ or system of organs.
3. Change in the position or form of some of the organs or parts of organs.
4. Deposits in or around the elementary parts of tissues, or changes of a degenerative or retrograde kind in them.

The object of prosecuting the anatomy of disease is, therefore, in the first instance, to institute a comparison between the known appearances or standard of health and an altered state of the parts. Such a comparison is, in the first instance, founded on an intimate knowledge of the doctrines stated at page 36.

Means and Instruments of Research.—To institute investigations such as those indicated at page 40, advantage must be taken of almost every branch of human knowledge. The methods of carrying on pathological research are therefore very varied, but may be shortly enumerated under the following heads:

1. The opening of dead bodies, to ascertain the condition of their

organs and tissues in all that relates to their structural, chemical, and physical properties (ROKITANSKY, HASSE, VIRCHOW).

2. Application of various instruments, such as the microscope, and of means to ascertain the absolute and specific weight of organs or parts, the relations, size, form, and colors of structures, and the like (QUEKETT, BENNETT, BEALE, PEACOCK, BOYD).

3. Application of chemical investigations to the diseased products (VOGEL, SIMON, DAY, LEBERT, GLUGE, BEALE, GARROD, CHRISTISON, PARKES, VIRCHOW, FRERICHs, GAIRDNER).

4. Application of statistics to determine various points of interest in reference to the nature, course, and complications of diseases (WM. FARR, GUY).

5. Means to preserve objects for further study by the microscope, or any other mode of examination (TULK, HENFREY, BEALE, QUEKETT, VAN DER KOLK, LOCKHART CLARKE).

6. Experiments instituted on living animals, and, in certain cases, on man, with the view of artificially producing a morbid condition. A careful study of such experiments by the previously mentioned means affords valuable information, for the causes in action are more under control than those which are spontaneously brought about by disease in the living body (BERNARD, HARLEY, PAVY, KUCHENMEISTER, ZENKER, and others).

The immediate object of such investigations is to obtain information regarding the material changes in the different parts of the body which accompany or produce morbid symptoms, and to connect these changes with symptoms and signs of disease during life. We thus learn how morbid products are formed at first and gradually perfected; and by combining these two kinds of knowledge we learn the relative connection of two orders of phenomena; namely, how the perverted properties, disordered actions, or altered structures give rise to perverted or impaired secretions; disordered and irregular motions; deranged, impeded, or interrupted functions. In other words, the "*order of invasion of disease-processes*" is learned from such investigations; and we are thereby taught how parts, once the seat of morbid change, return, by various processes of nutrition, growth, repair, or reproduction, to their normal condition.

The questions arising out of such investigations are, or ought to be, the first object of thought to the conscientious medical practitioner. It is his duty, from an attentive consideration of the signs and symptoms of disease, to form an idea, as accurate as possible, of the nature and extent of the morbid action or change which is going on, or which may be set up, in the tissues, organs, and fluids of the living body.

If, therefore, he does not avail himself of every means and instrument by which he can ascertain the existence of change in the dead body, and its alteration from some standard of health—if he does not embrace every opportunity of making post-mortem examinations—if he contents himself merely with observing signs or symptoms of disease, without witnessing the changes of structure, if any,

which may give rise to them—he can have little conscious satisfaction in the study of Medicine as a science, or in the practice of the healing art. In the words of Cruveilhier, he will, during his lifetime, “see many patients, but few diseases.” Such a practitioner is not to be trusted.

Various Forms of the Constituent Elements of Disease.

The histologist has now clearly ascertained the various simple organic forms which compose the textures in their normal state, and the mode in which these textures are arranged and combined so as to form the organs and systems which carry on the healthy functions of the body. The pathologist has made out (although with less completeness), by the methods of observation and experiment already indicated, the various simple organic forms which constitute the elements of those material changes whose phenomena of growth, decay, and varied change are associated with the manifestations of disease. By classifying and arranging these forms we obtain more clear ideas of lesions; and we ascertain that the material morbid processes follow, in their development, a very definite order of change, not yet in all cases determined with absolute certainty.

An anatomical investigation of morbid parts, conducted with the aid of the microscope and other instruments of research, shows that the material of which their substance is made up is of very various structure, sometimes combined in forms of one kind throughout, and sometimes varied by the development and combination of many elementary forms, more or less solid, soft, or fluid.

An analysis of the morbid material, carried as far as scientific means at present enable us, shows that the elementary conditions in which morbid products are found may be described as follows:

1. Fluid matter and hyaline substance, more or less soft.
2. Simple elementary forms of the nature of deposits, sometimes of a mineral or inorganic character; *e. g.*, (*a.*) amorphous granules; (*b.*) crystalline structures in a granular state.
3. Simple, but organized products capable of growth; *e. g.*, (*a.*) granules; (*b.*) compound corpuscles; (*c.*) simple cells; (*d.*) fibres.

The various appearances and conditions which these simple forms may assume in disease, as well as the functional states with which they are frequently associated, lead to a further enumeration and classification of morbid elementary products, as well as of more complex disease-processes, as below:

A.—MORBID ELEMENTARY PRODUCTS.

I. EXUDATIONS MORE OR LESS SOFT, SEMI-FLUID, OR FLUID, AND FORMED OF,—

a. Germinal plastic and formed material, which has sometimes been called *blastema*, *coagulable lymph*, *false membrane*, or *fibrine*, as seen adhering to free surfaces.

b. Aqueous matter, as seen in the morbid state termed "*dropsy*," and "*œdema*" of parts.

c. Gaseous exudations, as seen in the various forms of *pneumato-sis*.

II. EXUDATIONS MORE OR LESS CONSOLIDATED, AND CONSISTING OF,—

a. Molecular or granular material, from the 800th of a line to an immeasurably small size, and consisting chiefly of the simple forms of,—

(1.) Fatty molecules or granules.

(2.) Forms of an organic kind capable of growth, and invariably taking origin from a pre-existing structure.

(3.) Deposits of an inorganic kind, generally calcareous salts.

(4.) Pigment granules.

b. Coagulable compounds, resisting the action of most reagents, such as are seen in the elements of tubercle, scrofula, oleo-albuminous formations.

c. Exudations of a transitional nature, organized, which are capable of growth, which may become vascular, which grow from pre-existing structures, and which are composed of,—

(1.) Consolidated homogeneous material passing to

(2.) A fibriloid arrangement of the molecular or granular particles composing connective substance, and a subsequent formation of fibres in it or from it.

(3.) The formation of pyoid cells, and fibro-plastic or connective tissue cells, passing into fusiform cells and fibres as the material becomes consolidated.

(4.) The formation or exudation of fluid matter holding pus, or other more compound cells.

III. GROWTHS AND EXUDATIONS OF A MORE OR LESS SPECIFIC KIND.

a. Lymph of small-pox and cow-pox.

b. Matter of glanders, of malignant pustule, and of the plague.

c. Fluid of infecting chancre, and of some forms of secondary syphilitic lesions.

d. Material of tubercle and scrofula. (?)

e. Material of cancer.

f. The growth in Peyer's glands during typhoid fever.

g. The growth in Peyer's glands in cases of cholera.

h. Melanotic or pigmentary germs.

IV. MATERIAL OF A COMPLEX KIND.

a. Media of repair and reproduction of injured or lost parts—substance of granulations and cicatrices.

b. Hypertrophy of parts.

c. Tumors, $\left\{ \begin{array}{l} \text{innocent.} \\ \text{malignant.} \end{array} \right.$

d. Concretions.

V. PARASITIC FORMATIONS.

B.—COMPLEX VITAL PROCESSES WHOSE PHENOMENA, MORE OR LESS COMBINED, CONSTITUTE DISEASE.

1. *Fever*—the febrile state—*Pyrexia*.
2. *Inflammation*.
3. *Irritation*.
4. *Congestion*.
5. *Depression*.
6. *Atrophy*.
7. *Degeneration*.

Such a classification as the above is merely intended to bring before the student at a glance the variety of morbid material which is concerned in the expression of many of those phenomena seen in the course of disease, the distinctions made being mainly based on structural analysis.

While it is more properly the province of the anatomist to describe the MORBID ELEMENTARY PRODUCTS, it is the COMPLEX VITAL PROCESSES, WHOSE PHENOMENA, MORE OR LESS COMBINED, CONSTITUTE DISEASE, with which the Physician has more immediately to deal; and some of these complex states especially require notice here; namely, *Fever and Inflammation* and some forms of *Degeneration*.

CHAPTER VIII.

COMPLEX MORBID STATES.

SECTION I.—FEVER—*Pyrexia*.

Definition.—*A complex morbid state which accompanies many diseases as part of their phenomena, more or less constantly and regularly, but variously modified by the specific nature of the disease which it accompanies. It essentially consists in elevation of temperature, which must arise from an increased tissue-change and have its immediate cause in alterations of the nervous system (VIRCHOW, PARKES).*

Pathology of Fever and Phenomena which constitute the Febrile State.—In describing the nature of fever, the following statements are principally compiled from the Gulstonian Lectures of Dr. Parkes, delivered before the College of Physicians in 1855, and from a review by Dr. Jenner, “On the Proximate Cause of Fever,” in *The British and Foreign Medico-Chirurgical Review* for 1856. Knowing how difficult it is to convey an orthodox account of the nature of fever; fully impressed with the great importance of the subject; and believing, as Dr. Jenner has expressed, “that so consistent a theory of the nature of fever, and one so largely supported by facts, has not been placed before the profession as that developed by Dr. Parkes, I only hope I may be able to do it justice in the

attempt to lay it before the student of medicine in the following form. In the eloquent language of Dr. Parkes, 'I shall have to allude to inexplicable phenomena, to vast spaces still unfilled by solid facts, to spots unknown to observation, and to regions lighted only by the dim and treacherous ray of speculation.'

The practical object aimed at in the exposition about to be given, is to fix the scientific principles which ought to guide clinical investigation in determining the *Natural History* of fevers generally; and especially to define the differences which subsist among specific fevers; and so aid in determining the conditions under which fevers are generated or propagated—their development, course, or progress, and their deservescence.

"A hot skin, a quick pulse, intense thirst, scanty and high-colored urine," are phenomena common to many diseases; and when they are present it is said that the patient is *feverish*, or suffers from *fever* or *pyrexia*. There are some diseases in which such symptoms constitute the prominent, and almost the only appreciable phenomena, and which run a more or less definite course, without the necessary development of any constant local lesion. Such diseases have been emphatically termed "fevers," or sometimes *specific*, *primary*, or *idiopathic fevers*. When diseases marked by local lesions—such, for instance, as the local inflammations—are attended by the symptoms just stated, then the *pyrexia*, *fever*, or *feverish* symptoms which attend them, are said to be *secondary* or *symptomatic*; and the physician is accustomed, when he deals with such cases, to abstract the symptoms of fever from the other symptoms proper to the special affection. In other words, he prescribes for, and tries to cure the special affection, and not the *fever*, because he knows that when he has subdued the local disease the *fever* will subside. Not so, however, with the fever of a specific disease like *small-pox*, *typhus*, or *typhoid fever*: the physician cannot cure such a fever; but he may guide its course, by judicious management, as an experienced pilot may guide a ship and preserve it through a storm.

It is to the nature of *fever* considered in its abstract relations that the attention of the student is here directed, and not to any particular fever, such as *ague*, *typhus fever*, or the like. It is to *fever* in general that the following observations apply. It is to the *pyrexial symptoms* which are common to many diseases, such as to *small-pox*, *scarlatina*, *measles*, *typhus*, *ague*, *pneumonia*, *nephritis*, *meningitis*, and which, "like shadows to substance, are necessary to the very existence of such diseases, but yet are not, *per se*, any one of these diseases."

Galen defined fever as a preternatural heat—"Calor præter naturam." Subsequently many other additional clauses were added to this definition, such as "quick pulse," "turbid urine," and the like; but still the improved definition would not meet the requirements of every case; and now it is fully recognized that of all the clauses and phrases in the usual definitions of fever, "preternatural heat" is the only one whose accuracy is unimpeachable. In all cases, therefore, where fever is present, there are two points to be determined; namely,—(1.) *The amount of the preternatural heat by accurate*

measurement; (2.) *The amount of the tissue-change*, as represented by an estimation of the relative amount of all the excreta to the body weight.

It is the exact sequence of phenomena we desire to know in every case where pyrexia is present, as well as the meaning and co-relation of the phenomena: and usually symptoms sufficiently characteristic become developed and superadded to the febrile phenomena, by which the physician is able to define the specific nature of the disease or fever as a whole, and to say of this case or of that, "It is a *typhoid fever*," or "It is an *ague*," or "It is a *rheumatic fever*," or "a *pneumonia*," or "a *dysentery*," or any other form of illness where pyrexia is present, which we are able clinically to recognize. It is not very long since we were able to do this. Up till within a comparatively short time ago, the classification and diagnosis of "Fevers" was not such as to distinguish and separate their varied forms and varieties from each other. "Common continued fever," for example, was a comprehensive name which included many very different types of fever; and no means of observation have been of late so exactly discriminating, so as to distinguish one form of disease from another where fever coexists, as accurate observations on the temperature of the patient, determined by the thermometer. In acknowledging this great fact, it is important to observe that the absence of such exact observation, and the trusting to general signs alone, have hitherto led to great confusion—a confusion which has been most unjustifiably and unfortunately increased by a pernicious system—becoming too common—of naming "Fevers" from the place or locality where supposed varieties of fever have prevailed as epidemics; or by the use of local or provincial native names. For example, the Walcheren Fever, Levant Fever, Mediterranean Fever, Crimean Fever, Bulam Fever, African Fever, Fernando Po Fever, Lisbon Fever, Bengal Fever, Pucca Fever, Gall-sickness of the Netherlands, Hong Kong Fever, and other names not less barbarous, may be quoted. Except as matter of history, and as beacons to warn us from the danger to science, let these and such-like names be consigned to oblivion. With the exact means at the disposal of the physician as aids to diagnosis (and which are about to be described), every variety of illness where fever takes a part may be accurately distinguished, its type recognized, and its place fixed in nosology: or if it should be anomalous, its exact departure from the type may be not less accurately defined and described.

The phenomena which thus call for special investigation are those which are strictly related to the development and progress of the febrile state. They ought to be determined by clinical observation in all cases of disease where fever may be present. The facts to be ascertained are not less significant of the abatement, subsidence, or "*defervescence*" of the febrile state than of the advent of local lesions. The term "*defervescence*," in fever, is a comparatively new one in English pathology. It was first used by Professor Wunderlich, and subsequently adopted in this country by Dr. Parkes. It signifies the period during which the temperature of

the fevered body is declining to its normal amount from that intense degree of heat attained in the state of accession of the febrile phenomena. This "*defervescence*" may be sudden, when it is regarded as a "*crisis*;" or it may be gradual, and is then described as a "*lysis*"—the "insensible resolution" of the older authors; or it may be partly sudden and then slow, when it may be described as "*wave-like*," with gradual and sometimes regular alternations of high and low temperature, as Dr. Parkes was the first to point out (*The Composition of the Urine in Health and Disease*, p. 270).

The Usefulness of the Thermometer at the Bedside in the Diagnosis of Pyrexia.—One hundred and ten years ago (1754), Antonius de Haen, the first teacher of clinical medicine in the Hospital of Vienna, impressed his pupils with the necessity of attending to the temperature of the body in disease, *as measured by a thermometer*, instead of being judged of merely by the hand. He showed that even in the cold stage of ague, with the teeth chattering and the body shivering, the temperature of the blood is rapidly rising, although the pallid skin may really be colder than usual—its supply of blood being diminished by the contraction of the bloodvessels. He first demonstrated with *measured accuracy* how much the heat of the body is augmented under the influence of the febrile state; and when the crude appliances and the rough instruments of a hundred years ago are compared with the delicacy and refinements of "the instruments of precision" of the present day, it may be of interest now to observe how the progress of knowledge and the powers of modern research have not suffered the valuable pathological lessons to be lost sight of which are to be learned from the clinical use of the thermometer, as De Haen taught a hundred years ago. When the hand of the physician alone is used to judge of the temperature of a patient, or when the feelings of the patient are alone taken as a measure of his temperature, it can easily be understood how such kind of observation is extremely fallacious, doubtful, and unsatisfactory. The determination of the amount of heat in fever cases is stamped by a much more early appreciation of its importance and value than even since the time of De Haen; for, ever since the days of Hippocrates, the Physician and the Surgeon have been in the habit of applying the hand to the skin of the patient, to appreciate the presence of abnormal heat; but the practical application of the thermometer in place of the hand, while it is obviously a more accurate method, has never come into general use, mainly on account of the difficulty of getting instruments sufficiently sensitive and trustworthy—instruments, in fact, of sufficient precision. The time and trouble required to work with crude and inefficient instruments soon brought them into disuse and discredit; but now the instruments required may be obtained so delicate and accurate, and the time they take to apply them is so insignificant, that the student of medicine and the physician have no excuse for neglecting to use them. When it is remembered, also, that Galen's definition of fever is still the one whose accuracy remains not only unimpeachable, but fully demonstrated, and now recognized; that it describes fever to consist in "a preter-

natural heat,"—it is obviously absolutely essential that medical men should be able to measure this heat, and so learn the significance of such increase of temperature in every case of disease where fever may be present. The careful physician *counts the pulse and the respirations* in all cases of illness; it is now not less incumbent upon him *to measure the heat*. By means of a delicate thermometer he has in every case of fever an accurate measurer of its amount; and the student of medicine, as one of the earliest clinical lessons in hospital wards, should be taught to look to the excreta, and to the various physiological conditions of the patient, for the products of the metamorphosis of tissue equivalent to the amount of heat in each disease.

For physicians the thermometry of disease is practically important from two points of view, inasmuch as,—(1.) *The continuous daily use of the thermometer greatly facilitates the clinical recognition of diseases.* It aids the busy practitioner in coming to certain and safe conclusions; and so relieves him of much anxiety of mind in doubtful cases. (2.) *The use of the thermometer tends to elucidate the NATURAL HISTORY of all diseases where fever is present.*

It is proposed, therefore, to illustrate this subject under the following four heads:

I. *The Instruments, Methods, and Practical Rules for Observing and Recording the Temperature of the Human Body in Diseases where FEVER may be present.*

Animal heat has been determined in two ways; namely, either by the ordinary mercurial thermometer, or by the thermo-electric apparatus. The latter is able to indicate fractions of a degree, and in this respect surpasses the powers of the most delicate mercurial thermometers. MM. Becquerel and Breschet employed such an apparatus to determine the temperature of internal parts. The apparatus consisted of two wires, of different metals, soldered together, and having their free ends brought into communication with a *thermo-electric* multiplier, having an index showing 10ths of a degree. The wires being passed through different parts of the body (like acupuncture needles) indicate the temperature of the tissues at the point of contact of the two metals. For example—passing the wires an inch and a half into the calf of the leg, the temperature was found to be 98° Fahr., while at the depth of a third of an inch it was only 94° Fahr., showing some cooling of the body towards the surface compared with the interior. The superficial fascia of the biceps was nearly 3° Fahr. lower than the temperature of the muscle itself. But notwithstanding the greater delicacy of the *thermo-electric apparatus*, a sensitive mercurial thermometer, finely graduated, is the only instrument of practical usefulness, as yet, for ordinary clinical purposes; and for obvious reasons.

The Germans, who have been mainly instrumental in elucidating this subject, use thermometers which are graduated to Reaumur's or Centigrade scales—neither of which are in general use in this country.* These thermometers are cheap, and somewhat fragile,

* Mr. Griffin, of 119 Bunhill Row, has been able to import very good thermometers from Germany, graduated in Fahrenheit scale.

often differing also very much from each other. If German thermometers are used, it is therefore necessary to compare the thermometer used with a standard one, and note the differences *between every degree*. A thermometer is bad, and all but useless, if the differences between various degrees are unequal, but is quite serviceable if the same sum is to be added or subtracted for *each degree*. The price of such an instrument is moderate; therefore it need not be difficult nor expensive for a student to acquire a competent practical knowledge of "the thermometry of disease."

As it is necessary to have a good thermometer, with a uniform and correct scale, having a range from 88.2° to 110.7° Fahr., exhibiting also 5ths Fahr. of degrees, I have arranged, in a convenient wooden case (with the aid of Mr. Casella, the accurate and careful instrument-maker at 23 Hatton Gardens, London), two thermometers graduated in Fahrenheit degrees, especially for the use of medical men. Each box contains—

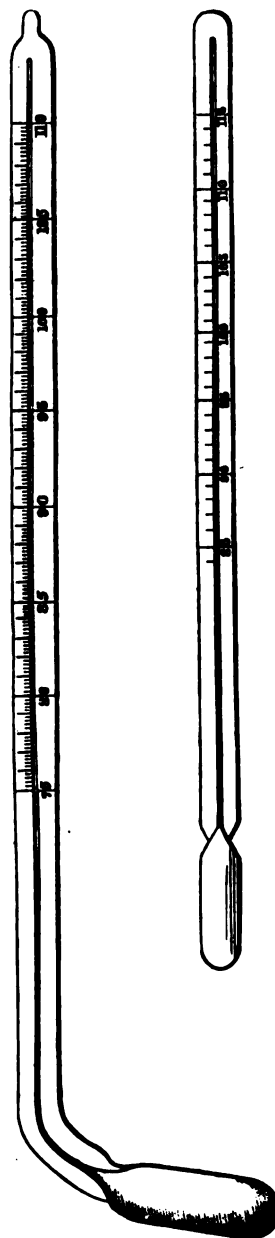
1. *An Ordinary but very Sensitive Thermometer*, Fig. A, made with a curve, in order that its bulb may be the more easily and perfectly fitted into the axilla, while the stem being carried upwards, renders the reading *in situ* more easy.

2. *A Straight Thermometer*, Fig. B, which, being a maximum self-registering one (known as "Phillip's maximum"), does not require to be read *in situ*, but may be removed from contact with the part, and read when convenient. Both thermometers are graduated up to at least 112° Fahr., and each degree is subdivided into *fifths*.

Directions for Use.—I. *The Curved Thermometer*, Fig. A. Its bulb must be well fitted into the arm-pit, being introduced below the fold of the skin covering the edge of the *pectoralis major* muscle, and so kept in close contact with the skin, completely covered and firmly surrounded by the soft parts. In very thin or very old persons this adjustment requires special care. The instrument must be retained *in situ* during a period of not less than *three minutes*; and the height of the mercury in the graduated stem must be read *while the thermometer is still*

FIG. A.

FIG. B.



One-half the real size.

undisturbed in the axilla, care being taken that the axis of vision falls perpendicularly on the column of mercury in the tube.

II. *The Straight Thermometer*, Fig. B. 1. Its index must be set *before* commencing to take an observation.

[N. B.—The index is the bit of mercury detached from THE COLUMN IN THE STEM OF THE INSTRUMENT.]

2. This index is to be set by bringing the bit of detached mercury down into the clear part of the stem, just below the lines which indicate the degrees. This is done by taking the bulb and stem of the instrument firmly in the hand, and then by a *single rapid swing of the arm* the index will come down the stem; and this *swing of the arm* must be repeated till the top of the index is at least below the lines which indicate the degrees.

3. After the index has thus been set, the bulb of the instrument may then be applied to the axilla, the surface of the belly, or between the thighs, or any part which is completely covered; and being retained in close apposition (by strapping, if necessary), with the surrounding soft parts for any length of time, the instrument is to be carefully and gently removed, when the *top of the index*—i. e., the end farthest from the bulb—will denote the *maximum temperature* during the period the instrument has been in perfect contact with the parts. The patient should have been at perfect rest in bed for at least one hour before observations on temperature are made.

III. *The Observations* ought to be continuous daily, and regularly taken at the same hour every day, throughout the whole period of sickness. The most useful periods for observation are—(1.) Between 7 and 9 o'clock in the morning; (2.) At noon; (3.) Between 5 and 7 o'clock in the evening; (4.) At midnight.

IV. In all observations of temperature the Pulse and the Respirations should be noted at the same time.

In the less important cases the physician may make at least one observation daily himself, and leave the others to the friends of the patient or the nurse, if either of them are sufficiently intelligent. This arrangement, however, is only justifiable so long as the observations correspond with those typical of the particular disease, and so long as they are in harmony with the other general signs of its course; but as soon as notable deviations from these conditions are observed, the physician ought to make the observations for himself. A difference of one-tenth R. = .225° Fahr., or of two-tenths R. = 2° Fahr., is not of any practical importance unless it is persistent.

In prolonged and severe cases an examination of the records of temperature made during the course of the disease will recall to mind the nature of the case more effectively than the most detailed written history. For this purpose it is desirable to exhibit on paper the daily thermometric changes, in the form of an angular line or a curve, and to note in the proper places short memoranda of the more important incidents or therapeutic events which have taken place during the progress of the disease. A second and third line may be added, illustrative of the changes in the pulse and the respiration.

In chronic cases, when febrile attacks and their concomitant dangers may be expected, as well as in acute cases, after return of the normal blood-heat *one* daily observation will be found sufficient. This single observation may be best made in the afternoon, or at that hour of the day in which generally some apparent change takes place.

It is advisable to induce nurses, friends, or other attendants on the sick (whenever they seem apt pupils), to make notes of any considerable excitement or restlessness, hot hands, increased heat of head, and to consult at once the thermometer. They may thus, perhaps, tranquillize the patient and his friends when the instrument does not indicate any material increase of heat; but the sudden appearance of any considerable increase of temperature would always be (as we have seen) a fact of vital importance.

It has been recommended by some to place the thermometer under the tongue, as the best place. On the contrary, the cavity of the mouth is the worst place in which the thermometer can be put, because the temperature there is continually varying according to the quantity and temperature of the air used in respiration; and if the atmosphere is cold, and deep inspirations are made, large differences may be observed, compared with the temperature in the axilla. Therefore it is necessary not to trust to observations made with the thermometer in the mouth.

In cases which do not require the most rigorous and extreme accuracy (as cases observed for the sake of scientific information require), *three* to *five* minutes is found quite sufficient in private practice for the application of the thermometer. The simplest and most convenient way is to heat the instrument before inserting it into the patient's axilla, just as the surgeon heats the catheter before he introduces it into the urethra. This may be done by holding the thermometer in the warm hand, or into water heated to a temperature of nearly 30° R. = 99.5° Fahr.: and, after the instrument is properly placed, be satisfied if *two* observations at intervals of *one* to *two* minutes give exactly the same result.

The rapidity with which the mercurial column rises depends on the degree of temperature present. The rapidity of the rise of temperature ought to be noted, as well as the maximum height. If the temperature be above the normal standard, a sensitive thermometer will indicate that fact within the first minute; and as the quickness of the rising depends upon the existing temperature, the physician is able, after some experience in the use of a particular instrument, to form an approximate judgment of the amount of rising of temperature to be expected in any particular case from the slowness or rapidity of the rise of the mercury after half a minute.

II. *Fluctuations of Temperature within the Limits of HEALTH; and the Co-relation of the Animal Heat with the Pulse and the Respiration.*

Several observers in Germany, France, England, and the Tropics, have now determined these fluctuations with great accuracy, so that ample and sufficient data are on record to furnish a standard for comparison in cases of disease.

The temperature of the body is the result of the opposing action

of two factors: 1st, Of development of heat from the chemical changes of the food, and by the conversion of mechanical force into heat, or by direct absorption from without; and 2d, and opposed to this, Of evaporation from the surface of the body, which regulates internal heat (Parkes's *Hygiène*, p. 432).

With reference to the normal range of temperature, our most trustworthy information is mainly due to Valentin and Traube, in Germany; to Edwards, Becquerel, Breschet, and Bernard, in France; and to Dr. John Davy, in England and the Tropics. All agree in stating that the ranges of temperature vary in different parts of the human body; but, as a general practical result, it is equally agreed that in temperate regions the normal temperature *at completely sheltered parts of the surface* of the human body amounts to 98.4° Fahr., or a few tenths more or less; and a rising above 99.5°, or a depression below 97.3° Fahr., are sure signs of *some kind of disease, if the increase or depression is persistent.*

Valentin proved by many experiments that all warm-blooded animals surrounded by an atmosphere of 50° Fahr. to 68° Fahr. have a temperature of about 99.5° in the back of the mouth, the rectum, or other accessible *internal parts*; and at *completely sheltered parts of the surface* it is a degree lower—namely, 98.4° or .5°. But the most common temperature is about 97.5°, according to Dr. Compton's more recent observations. Dogs have a temperature similar to that of men; so that a knowledge of thermometry in the diseases of animals will prove not less valuable in veterinary pathology than in human, and perhaps more so, inasmuch as such animals are deprived of speech to express their feeling.

The observations of Dr. John Davy, originally communicated to the Royal Society, are the most numerous and extensive in detail on record, which indicate the fluctuations of temperature within the limits of health. An abstract is given in the following table:

**FLUCTUATIONS OF TEMPERATURE WITHIN THE LIMITS OF
HEALTH (DAVY).**

I.—IN TEMPERATE REGIONS (ENGLAND).

Period of Day.	Temperature of Body.	Pulse per Minute.	Respiration per Minute.	Temperature of Room.
Morning, 7-8,	98.74° Fahr.	58	16	50.9° Fahr.
Afternoon, 3-4,	98.52° "	55	15	54.7° "
Midnight, 12,	97.92° "	55	15	62° "
II.—IN TROPICAL REGIONS (BARBADOS).				
Morning, . . 6	98.07° Fahr.	54	14	76.7° Fahr.
Afternoon, . . 2	98.9° "	56	15	83.6° "
Night, . . 10-11	99.0° "	60	15	79.8° "

The general result may be stated as follows:

1. IN TEMPERATE CLIMATES the *Maximum temperature* is in the

early morning after waking: it fluctuates till nightfall, and is lowest about midnight; average difference, 0.82° .

2. IN TROPICAL REGIONS the *Minimum temperature* is in the early morning after waking: it fluctuates, and is highest during the day.

3. *Average temperature* throughout the year, 98.4° Fahr.,—the temperature of the air averaging 55.5° Fahr.

The observations of Dr. Davy and of Edwards have shown that the amount of animal heat may be considerably altered by a number of collateral circumstances. But the great distinction between these alterations of temperature in health, and those which are the result of disease, is, that these variations are generally temporary, and within narrow limits—amounting to mere fractions of a degree—rarely more than from 1.8° Fahr. to 3.6° Fahr. (VALENTIN and DAVY), whereas those which are due to disease are persistent so long as the disease exists.

The following are the collateral circumstances which mainly influence animal heat in our daily life, and which require to be remembered in order that erroneous conclusions may not be drawn: (1.) *Active exercise* (not carried to the extent of exhausting fatigue) raises the temperature proportionally to the degree of muscular exertion made. (2.) *Exposure to cold* without exercise lowers the temperature. (3.) *Sustained mental exertion* reduces temperature about half a degree. (4.) The amount of heat is also at first reduced after a full meal and the use of alcohol; but it rises again as digestion advances. (5.) There are diurnal fluctuations capable of being thus determined. (6.) The temperature of the body rises with the temperature of the air; and sudden transitions from a cold to a hot climate induce a feverish state marked by increase of temperature, accelerated action of the heart, and quickened respiration, especially on bodily exertion. (7.) The average temperature within the tropics is nearly 1° Fahr. higher than in temperate regions. (8.) The temperature is more readily and rapidly affected—more sensitive, so to speak—than either the pulse or the respiration; and this is especially the case in disease.

The amount of abnormal increase of temperature is usually proportionate to the degree of frequency of the pulse, and to the other signs of general disease. Yet such congruity of phenomena is sometimes in part or wholly absent or incomplete; and in the cases in which a disproportion or incongruity exists between the increase of temperature and the pulse or other febrile phenomena, it is the accurate measurement of the temperature which is most of all to be relied upon. As a general rule the co-relation of pulse and temperature may be stated as follows, namely: "*An increase of temperature of ONE DEGREE above 98° Fahr. corresponds with an increase of TEN beats of the pulse per minute, as in the following table:*"

Temperature of	98°	.	.	.	Corresponds with a pulse of	60
"	99°	.	.	.	"	70
"	100°	.	.	.	"	80
"	101°	.	.	.	"	90
"	102°	.	.	.	"	100
"	103°	.	.	.	"	110
"	104°	.	.	.	"	120
"	105°	.	.	.	"	130
"	106°	.	.	.	"	140

III. *Ranges of Temperature in Disease.*

Having satisfied ourselves as to the delicacy and accuracy of the thermometer, and obtained a standard for comparison, we are prepared to appreciate the range of temperature in febrile disease as measured by such an accurate instrument.

The maintenance of a normal temperature, within the limited fluctuations just noticed, under all these varying influences, gives a complete assurance of the absence of anything beyond local and unimportant disturbances; and, long before the subject was worked out so thoroughly as it has been, it was often casually observed that any acute disease, however slight, elevates abnormally the temperature or animal heat; "and its undue degree of elevation (as Dr. Davy clearly enunciated) is some criterion of the intensity of the diseased action" (*Physio. Researches*, vol. i, p. 56). In short, it is now placed beyond a doubt by the observations of Gierse, Roger, Valentin, Von Bærensprung, Wunderlich, Friedlander, Virchow, Traube, Jockmann, Greisinger, Billroth, and others, in Germany; by MM. Becquerel, Breschet, and Bernard, in France; by Parkes, Jenner, and Ringer, in this country, that while this preternatural heat varies in amount in different diseases in different persons, and at different times of the same day, it is this preternatural heat which is the essential symptom in fever, which proves fever to be present, and which exists to the extent of 4°, 6°, or even 8° Fahr. over the natural limits of health (which averages 98° Fahr.), and must be judged of by the temperature in the axilla, as indicated by the thermometer. This preternatural heat is never absent in fever, and without it fever cannot be said to exist. Rigor, which is also sometimes present, is a mere peripheric phenomenon, and the coldness of the skin a subjective sensation, produced by the state of the peripheral nerves, and is not due to any actual decline of temperature. "While the outer parts feel cold to the bystander, the inner parts are abnormally warm. While the outer parts freeze, the inner burn" (VIRCHOW, PARKES, JENNER).

There are many cases now on record in which the physician, without thermometric observation, does not appreciate the existence of fever or of danger. Wunderlich gives numerous examples of this; but long before he brought this subject so forcibly to the notice of medical men, we have the testimony of Dr. John Davy in this country, given quite incidentally, and therefore all the more valuable as an unbiassed testimony of the usefulness of the thermometer in detecting latent disease not otherwise indicated by general symptoms. When Dr. Davy was collecting his extensive observations on the normal temperature of the body, he was surprised to find that one person exhibited for many weeks a persistent temperature of 104° Fahr. This person was a lunatic soldier, and Dr. Davy remembered that the insane do not seem to suffer from cold nor heat like ordinary individuals, and that there are certain organic lesions which are apt to occur in them, unaccompanied by the usual symptoms. For example, tubercle and cavities of the lungs occur without cough or difficult breathing; and although no warning nor any indication may be given, the disease runs its

course, terminating in death, as certainly and as rapidly as if indicated by the ordinary train of symptoms. Discovering then, as it were by accident, that the temperature in this lunatic was as high as 104.5° Fahr., and that his pulse was rapid, Dr. Davy's attention was more particularly aroused; and although the man made no complaint, but had a good appetite, with his digestive functions, so far as was known, acting well, yet disease of the lungs was thus discovered. The lunatic died in a month, of acute tuberculosis, not otherwise expressed by symptoms beyond the great, persistent, and continuous elevation of temperature thus incidentally noticed. There were ulcers of the *larynx* found after death, but there had been no affection of the voice; there were *vomicæ* and *tubercles* in the *lungs*, but there had been no cough; there were *ulcerations* of the *intestines*, but there had been no diarrhœa; there was disease of the *testes*, *vesiculæ seminales*, and *prostate*, of a severe kind, but these lesions had been equally latent during life, except hardening and enlargement of the testicle without pain,—all which conditions were only casually observed.

In this very instructive case, a temperature of *six degrees* Fahr. above the normal standard was the earliest indication of disease (*Researches, Physiological and Anatomical*, vol. i, p. 206).

But it is mainly to Wunderlich, the Professor of Medicine in Leipzig, that we are indebted for an elaborate exposition and persevering advocacy of the usefulness of daily records of the temperature of fever patients, and the constant employment of the thermometer as a means of diagnosis at the bedside. On this subject he has written much, from an extensive experience, embracing at least half a million exact thermometric observations, following the continuous progress of individual diseases, the results of which he has compared in more than 5000 patients. He constantly employs the thermometer in his private practice, and bears unqualified testimony to its sterling value in the early detection of disease, and as often furnishing an important guide to treatment. When the physician once becomes accustomed to the investigation of disease by the thermometer, he regards its daily employment as indispensable, for it imparts a certainty to his observations, attainable by no natural penetration, and which no other method of investigation can convey (*Medical Times and Gazette*, June 19, 1858, and September 28, 1861).

More detailed results are published by the assistants or pupils of Wunderlich, in the *Archives für Physiologische Heilkunde*, 1860, p. 385, and 1861, p. 433; and the principal conclusions have been summed up by Wunderlich himself, in his *Handbuch der Pathologie*. From these sources the information given in this and former editions of this text-book was originally compiled.

Wunderlich gives some striking instances of disease being indicated by thermometric observation before it could be detected by any other means:

In ague, several hours previous to the paroxysm, the temperature of the trunk of the patient's body begins to rise; and when the disease seems to have disappeared, an increase of temperature may be

detected periodically, unaccompanied by any other symptom. So long as this periodic rise of temperature continues, the patient is only apparently, but not really cured.

In typhoid fever, during the exacerbations especially, the rise of temperature or its abnormal fall will indicate what is about to happen three or even four days before any change in the pulse, or other sign of mischief, has been observed. A sudden and marked reduction of temperature has thus denoted hemorrhage from the sloughs of Peyer's patches in typhoid fever, several days before it appeared in the stools. A case of this kind is recorded by Dr. Parkes. It occurred in a female twenty-five years of age. Diarrhœa was considerable, and blood was largely passed in fluid stools the night before the 17th day of the fever. On the morning of that day the temperature was as low as 93° Fahr., rising in the evening to 101° Fahr.

It is rare, however, that a definite diagnosis or prognosis can be based on a single observation; but sometimes it may be sufficient, as in the following instances:

When the temperature is increased beyond 95.5° it merely shows that the individual is ill, and suffering from some disease, and that when considerably raised, as with a temperature of 101° to 105°, the febrile phenomena are severe; that when a great height is reached, as at temperatures above 105° Fahr., the patient is in imminent danger; and that with a rising temperature above 106° Fahr., to 108° or 109° Fahr., a fatal issue may almost without doubt be expected in a comparatively short time. The highest temperatures before death have been observed in cases of scarlet fever and of tetanus.

A definitive diagnosis may also be based on a single observation, under the following circumstances:

A person who yesterday was healthy, but exhibits this morning a temperature above 104° Fahr., is almost certainly the subject of an attack of ephemeral fever or of ague; and should the temperature rise up to or beyond 106.3° Fahr., the case will certainly turn out one of ague, or some other form of malarious fever.

A girl eighteen years of age, supposed to be suffering from hysteria, but simulating a case of cerebro-spinal meningitis. A temperature of 103.5° confirmed the diagnosis of meningitis and negatived that of hysteria. The case terminated fatally (COMPTON). Again, in a patient whose temperature rises during the first day of illness up to 106° Fahr., it is certain he does not suffer from typhus nor typhoid; and of a patient who exhibits the general typical signs of pneumonia, but whose temperature never reaches 101.7° Fahr., it may be safely concluded that no soft infiltrating exudation is present in the lung.

Again, if a patient suffer from measles, and retains a high temperature after the eruption has faded, it may be concluded that some complicating disturbance is present.

Single observations with other means of diagnosis will often determine whether the disease is one of danger or not.

In typhoid fever a temperature which does not exceed on any

evening 103.5° Fahr. indicates a probably mild course of the fever—and especially if the increase of temperature takes place moderately, towards the beginning of the second week. A temperature of 105° Fahr. in the evening, or of 104° Fahr. in the morning, shows that the attack is a severe one, and forebodes danger during the third week; on the other hand, a temperature of 101.7° Fahr. and below, in the morning, indicates a very mild attack, or the commencement of convalescence. In pneumonia a temperature of 104° and upwards indicates a severe attack. In acute rheumatism a temperature of 104° is always an alarming symptom, foreboding danger, or some complication such as pericardial inflammation. In a case of jaundice otherwise mild, an increase of temperature indicates a pernicious turn. In a puerperal female an increase of temperature indicates approaching pelvic inflammation. In tuberculosis an increase of temperature shows that the disease is advancing, or that untoward complications are setting in.

In short, a fever temperature of 104° to 105° Fahr. in any disease indicates that its progress is not checked, and that complications may still occur.

But it is by continuous daily observations that the most important results have been arrived at, especially in the hands of Wunderlich, Greisinger, Traube, Billroth, Parkes, Jones of Augusta, Ringer, and others who are now working most actively in this field of labor.

Certain febrile diseases have been found to have typical ranges or daily fluctuations of temperature throughout their course. In pure unmixed and uncomplicated cases this is found to be so constant that the differential diagnosis may be established by accurate observation of the temperature continuously from day to day. This has now been determined, especially in cases of malarious fever, typhus, typhoid fever, small-pox, scarlatina, measles, rheumatism, pyæmia, pneumonia, acute tuberculosis. In each of these diseases the temperature is one of the most certain (although not the only) means for determining the real state of the patient as regards morbid disturbances or complications, and a careful observation of temperature from day to day is indispensable for judging as to the prognosis. Frequently it affords the best and ultimate means of deciding in doubtful cases, and often it is the best corrective of a too hasty conclusion: for example, the characteristic variations of the temperature in a typical case of enteric, intestinal, or typhoid fever, are of such a kind that they are not found in any other disease. Intestinal catarrh, severe forms of pneumonia, malarious fever, acute tuberculosis, meningitis, some stages of Bright's disease, may each simulate typhoid fever, and may exhibit some of its most characteristic symptoms; but observation with the thermometer as to the patient's temperature from day to day, will at once, or after a very few days, establish the distinction with certainty.

In the course of many diseases, whose diagnosis has been accurately determined, if the temperature departs from its normal or typical range, the thermometer will furnish the best and the earliest

indication of any untoward event, such as the additional development of disease, or other visceral complications in its course.

When once the typical range of temperature (*normal*, as it were, of the particular disease) is determined, a basis is laid for appreciating irregularities or complications in its course in particular cases. For example, a patient exhibits symptoms of fever of the typhoid type, but during the progress of the first week his temperature becomes normal, for however short a space of time;—the occurrence of this event proves that the fever is not what it was supposed to be. Again, a patient may suffer from all the general symptoms of incipient pneumonia; but there still is a doubt as to whether infarction of the lung has taken place. The sputa being suppressed, or not procurable, does not assist the diagnosis. If, however, the temperature is found to be normal, it is certain that no croupous exudation has taken place in the lung, and that there is no pneumonia. Again, if a tuberculous patient has a sudden attack of hæmoptysis, and if the temperature of his body is normal during and subsequent to the attack, no reactive pneumonia, nor any exacerbation of the tuberculous exudation need be expected. This is a new field open for investigation in cases of phthisis.

Again: In all cases of convalescence, so long as the defervescence proceeds regularly as measured by the temperature, no relapses need be feared: on the other hand, delayed defervescence in pneumonia, the persistence of a high evening temperature in typhus or typhoid fever, or the exanthemata, and the incomplete attainment of normal temperature in convalescence, are signs of great significance. They indicate incomplete recovery, supervention of other diseases, unfavorable changes in the products of disease, or the continuance of other sources of disturbance requiring to be carefully examined into. The onset of even a slight elevation of temperature during convalescence is a warning to exercise careful watching over the patient, and especially for the maintenance of a due control over his diet and actions.

Continuous daily observations by the thermometer thus teach the typical laws of particular forms of fever, and supply the grounds or basis by which it is determined whether any individual case is progressing as it ought to do. Such knowledge can only be acquired by repeated observation of numerous cases; and deviations from the normal temperature in certain diseases are stable in proportion to the typical character and full development of the particular disease. But even in such diseases we may have an increase or decrease of temperature proper to the disease brought about by *accidental* influences. Such instability, however, is only temporary, and of short duration, when the *accidental* influences act but *transitorily*. For example, the temperature proper to the disease may be lowered under the influence of a profound sleep, bleeding, epistaxis, the relief of constipation or of the retention of urine, and the like; or it may be raised after excitement of a mental kind. But any such alterations, unless they are dependent upon a change in the disease-process itself, will become effaced after twelve or twenty-four hours at the most, when the temperature again resumes the typical

character diagnostic of the particular disease. In continued fevers the temperature is generally less high in the morning than in the evening.

Stability of temperature from morning to evening is a good sign; on the other hand, if the temperature remains stable from evening till the morning, it is a sign that the patient is getting or will get worse.

When the temperature begins to fall from the evening to the morning, it is a sure sign of improvement; on the other hand, a rise of temperature from the evening till the morning is a sign of his getting worse.

When it is found, in a bad case of typhoid fever, that some morning about the third week the temperature has fallen to 99.5° , the reparative stage has begun—the healing of Peyer's patches; and when a similar fall of temperature is observed in the evening, convalescence has commenced.

In pneumonia, when a marked fall of temperature occurs in the evening, the period of crisis has arrived.

In measles, when the maximum severity of the eruptive stage has been reached, the temperature falls.

A sinking from a considerable height down to a normal temperature suddenly (within twenty-four hours), occurs in a few exanthemata,—measles, variola, rarely in pneumonia, typhus, and pyæmia.

In tuberculosis, especially in its acute form, the persistent maintenance of a uniformly high temperature will alone show that no arrest in the progress of the disease has occurred.

The correlation of pulse, respiration, and temperature is of great importance to be determined in many acute diseases; and especially in pneumonia, if the mean of the temperature is not above 104° Fahr., and that of the pulse is not above 120 in a minute, and the mean of the respirations not over 40 in the same time, the case must be considered a slight one; and if the patient is otherwise healthy, he will surely begin to get well in from eight to twelve days, without any medical treatment beyond attention to antiphlogistic regimen.

Convalescence is known to commence when the disease-process ends; and this precise point can only be fixed by continuous thermometric observation. The morbid process does not end till the normal temperature of the body returns, and maintains itself unchanged through all periods of the day and night.

Regularly continuous observations of the temperature exhibit the precise point at which the disease-process terminates, and the degree of its complete development. When this point has been determined on, a retrospective view may be taken of the character of the disease, as to the purity of its typical form or its complexity, and a prognosis may be hazarded as to the probability or doubtfulness of recovery. The morbid process has not terminated till the normal temperature of the body returns, and remains unchanged in the evenings and throughout all periods of the day. The transition from the febrile state into defervescence is either slow (lysis) or rapid (crisis). A regularly continuous defervescence is always a sure sign of convalescence. Its occurrence will save other investi-

gations: irregular defervescence, on the contrary, indicates a disturbed and protracted course of convalescence, which requires careful watching and judicious nursing.

It is of practical importance to know that the fall of temperature during the period of recovery, in cases of considerable morning remissions, as well as in those of continued defervescence, may be abnormally large, and sink as low as 28° R. = 95° Fahr., or even lower. Such events constitute collapses during defervescence, which must be counteracted by artificial heat, the administration of warm drinks, or even of such stimulants as wine or camphor, unless some unexpected new danger should interfere with an otherwise favorable course of the disease.

During convalescence the recurrence of a high temperature is generally the first sign of an approaching relapse, or the onset of a new disease, the characteristic symptoms of which it may precede by several days. The persistence of even an inconsiderable degree of abnormal temperature after apparent return to health, is a certain, and frequently for a long time the only, sign of incomplete recovery, or the existence of some lingering secondary disease. Thus the temperature should be closely watched during convalescence; and the thermometer should be applied every alternate evening at the very least. As long as the temperature remains normal, nothing need be feared; but every rise of temperature should act as a warning. It may be due to mere error in diet, or to leaving bed too early; but in such cases the temperature soon sinks again, on greater precautions being taken.

Regularly continuous observations on the temperature alone, or in connection with other symptoms, may enable the physician to predict a fatal issue with certainty, or the probably near approach of death. On this point one of two conditions may be observed,—(1.) The temperature may rise continuously and considerably above 33° R. = 106.2° Fahr., when it is a bad sign; or it may even reach 34° R. = 110° Fahr., when a fatal issue is almost certain; and it not unfrequently happens that, after the apparent occurrence of death, the temperature still continues to rise one or two-tenths R. = $.225^{\circ}$ or 2° Fahr., the cooling of the body taking place very slowly. Wunderlich records a case of spontaneous or rheumatic tetanus, in which the temperature exceeded the maximum that has ever yet been observed in any disease. The heat only began to increase within the last twenty-four hours before death; but the other symptoms before that time had been very violent, the respirations being accelerated, and the pulse at 102. During the night previous to death the temperature suddenly rose 3.3° Fahr., while the velocity of the pulse and the frequency of the respirations diminished, and the other symptoms did not increase in severity. Shortly before death the heat rose to 110.75° Fahr., the pulse being then at 180; and at the moment of death the thermometer was at 112.5° Fahr. After death the temperature still rose, and was found to be 113.8° Fahr. an hour after the fatal event. It then slowly diminished; and thirteen and a half hours after death the temperature had not yet fallen to the normal average of the living body.

(2.) The temperature may become more or less moderated, while the pulse is increased in frequency, and the other symptoms become more and more threatening. Such diminution of temperature, amidst conditions which do not harmonize with it, must be regarded as a pretty certain sign of approaching dissolution.

But, on the other hand, there are cases in which the observation of the temperature yields the most favorable signs for prognosis. For example, when it is found, in a bad case of *typhoid fever*, that the temperature has fallen some morning to $30^{\circ} \text{ R.} = 99.5^{\circ} \text{ Fahr.}$, we know that the reparative stage is entered upon; and when a similar fall of temperature is observed in the evening, convalescence has commenced. In *pneumonia*, when a marked fall of temperature occurs in the evening, it shows that the period of crisis has arrived. When the temperature falls in *measles*, the maximum severity of the eruptive stage has been reached; and when, in the first stage of *variola*, we observe a quick return to the normal temperature, we may feel certain that a slight form of the disease, free from danger, is likely to ensue.

A decrease of temperature below the normal heat is rare. It happens sometimes transitorily, announcing thereby a favorable crisis, by preceding the return to a normal temperature. It is also met with sometimes during the morning remission of *remittent fever*; also during the *apyrexia* of *intermittents*; in acute collapse, preceded or not by fever; in chronic wasting diseases; and sometimes, also, on the approach of death, especially in typhus fever.

A remarkable inequality in the distribution of the temperature over different parts of the body (face, hands, feet, etc.) may occur during the shivering preceding fever, in collapse, and in the agony of approaching dissolution. Sometimes, also, such unequal distribution may occur in disorders of the chest and abdomen, in some local skin diseases, and in partial paralysis. This fact is not of importance or utility, for diagnosis or prognosis; but it requires to be known, in order that erroneous conclusions may not be drawn.

IV. *Of the Ranges of Temperature in Diseases where Fever is present, as related to the Amount of the Excreta.*

The particular degree of heat and the waste in every febrile disease are represented by—*something*. The physician sees the fevered patient wasting before his eyes. Every tissue is wasting, and, in correlation with the excessive generation of heat, how is this waste expressed? As a rule, it is expressed by the amount of *excreta*.

To Dr. Parkes in this country, to Dr. Jones of Augusta (in cases of malarious fever), to Virchow and Wunderlich in Germany, is the merit mainly due of having demonstrated, by clinical and experimental observation, that "the morbid development of heat, as measured by a thermometer, is associated in some cases with MORE abundant, in other instances with LESS abundant *excreta* from the body than in health; that the temperature and the amount of the excretions bear some undetermined relation to each other; and that the loss of weight of the patient is due to increased and rapid elimination of material with increased tissue-change, associated with the increase of temperature."

So far as physiological facts have elucidated the normal generation of heat in the healthy body, so far has the abnormal generation of heat essential to the *febrile* state been clearly made out. In health the normal temperature produced by chemical change in the body, is represented in the excretions by so much *urea, sulphuric acid, carbonic acid, excretive volatile acids* of the skin, and the like; but in the febrile body a higher temperature is represented in the excretions, in some cases by a larger, and in others by a smaller quantity of *urea, sulphuric acid, and probably carbonic acid** (PARKES).

The most opposite statements have thus been made regarding the amounts of the excretions in fever, compared with the quantity excreted in health; and at present many excellent observers hold that these excretions are always, and of necessity, increased; others, no less exact, affirm that they are invariably, or almost always, diminished. Such discrepancy of statement is due, in the first instance, to the difficulty of collecting and measuring exactly the amount of all the excretions. "Two of the excretions, the cutaneous and the pulmonary, cannot be collected and measured with anything like the accuracy necessary in such an inquiry: even in health, such an inquiry is difficult, and in fever it is almost impossible." By careful and accurate observation at the bedside, however, Dr. Parkes has been able to obtain very close approximative data to found his conclusions upon, relative to the increase or diminution of the excretions. He assumes that when the respirations are not quickened (*i. e.*, about eighteen times in a minute, or about one act of respiration for every four beats of the pulse), and when the skin is not evidently sweating, the excretions by these two organs are not increased; and, on the other hand, an increased excretion by these organs may reasonably be inferred, if the exercise of their function is unusually active, and if there are tolerably copious perspirations. The other two excretions, namely, the urine and intestinal discharges, can be measured with accuracy, and the urine in particular is a valuable index of the metamorphoses of tissue. The *urea* alone represents two-thirds of the whole quantity of *nitrogen* which passes off; the *sulphuric acid* (the sulphates of the food being

* In the study of special diseases, the student ought frequently to estimate the quantity of *excreta* by the urine, as one of the best methods for enabling him to appreciate the changes which go on in the body during disease. To aid him in prosecuting such researches, he is recommended to consult the work of Dr. Parkes, *On the Composition of the Urine*, and to follow the directions given in the *Examination of the Urine*, towards the end of the second volume of this text-book, for obtaining quantitative results by the volumetric method.

Average quantity of urine passed in twenty-four hours, .	52½ to 56 ounces.
Average amounts of solids	945 grains.
" " <i>Urea</i>	512 "
" " <i>Chlorine</i>	126.76 "
" " <i>Free Acid</i>	33 "
" " <i>Phosphoric Acid</i>	48.90 "
" " <i>Sulphuric Acid</i>	31.11 "
" " <i>Uric Acid</i>	8.5 "
Specific gravity.	10.20

accounted for) represents almost entirely the oxidation of sulphur; and the oxidized phosphorus of the body passes out in great measure, though not altogether, as urinary phosphoric acid. Therefore a careful examination of the urine, and of the intestinal discharges, with an approximative estimate of the pulmonary and cutaneous excretions, give sufficiently extensive and accurate materials for the question at issue.

The products excreted are thus of such a kind as to be eliminated, some by *the lungs*, some by *the skin*, some by *the bowels*, and some by *the kidneys*, and rarely by two or more modes of excretion—for when the discharges from the *skin* or *bowels* are profuse, those by the *kidneys* are deficient, as in the last two cases recorded in the following Table I, in which the augmented excretions are printed in italics. The facts thus so carefully observed by Dr. Parkes, confirmed by Alfred Vogel, Heller, and others (but chiefly in regard to the excretion of urea only), justify the conclusion—“*That increase of temperature may be attended with increased elimination; and therefore presumably with increased tissue-change.*”

TABLE I.—ABSTRACT OF CASES OBSERVED BY DR. PARKES IN WHICH SOME OF THE EXCRETIONS ARE INCREASED IN CONSEQUENCE OF THE FEBRILE STATE.

Disease.	Average Temperature above 98°.	Condition of Pulmonic Function.	Condition of Cutaneous Function or Skin.	Condition of Intestinal Function.	Condition of Urinary Excretion.
Rheumatism.	Fahr. 8°.	Not noted.	<i>Sweating profusely.</i>	Discharge as usual.	Solid matter excreted greater than in health by 100 grs., and due to urea and sulphuric acid.
Rheumatism.	Fahr. 8°.	Not noted.	<i>Sweating profusely.</i>	Discharge not diminished.	<i>Solid matters excreted greater than in health by 200 grs., and due to urea and sulphuric acid.</i>
Typhoid Fever.	Several degrees.	<i>Rapid.</i>	<i>Moist.</i>	Not increased.	Increased by 60 grains.
Erysipelas of Head and Face.		<i>Quick.</i>	<i>Moist.</i>	Unaltered.	Considerably augmented.
Febricula.	Fahr. 8°.	Normal.	<i>Enormously augmented.</i>	Confined.	Solids less than normal by 91 grains.
Typhoid.	Fahr. 8°.	Not noted	<i>Sweating and Sudamina.</i>	<i>Diarrhoea profuse.</i>	Solids less by 78 grains.

TABLE II.—CASES OBSERVED BY DR. PARKES IN WHICH THERE WAS DIMINUTION OF THE EXCRETIONS.

Disease.	Average Temperature above 98°.	Condition of Pulmonic Function.	Condition of Cutaneous Function or Skin.	Condition of Intestinal Function.	Condition of Urinary Excretion.
Bronchitis of both lungs.	Fahr. 2.6°.	20 Respirations per minute.	Not increased.	Not increased.	Less by 112 grains.
Pneumonia acute sthenic.	Fahr. 5°.	80 per minute; expectoration scanty.	Slightly moist.	Confined.	Less by 220 grains.
Typhoid Fever.	Considerable.		No sweating.	No diarrhoea.	Below normal amount.
Acute Rheumatism.	Fahr. 4°.	Tranquil breathing.	Inconsiderable.	Bowels quiet.	Very small amount of urinary solids.

The abstract given in Table II shows that another conclusion is equally legitimate, namely—"That the products of metamorphoses, as judged of by the excreta, may be diminished in febrile cases; and these apparently discordant statements are capable of being explained in various ways. In the first place, it is evident that more chemical change may go in the body than is represented by the excreta. The metamorphosis of blood or of tissues may not be carried to the point of forming those principles which can alone pass through the eliminating organs. A vast amount of imperfectly organized compounds may be formed and retained in the system, circulating with the blood or thrown upon certain organs." *Thus there may be increased metamorphosis with lessened elimination.* Several pathological facts point to such a conclusion.

1. It is in such febrile cases, with diminished excreta, that, at a later period of the disease, copious discharges from one or other of the eliminating organs occur. Thus, in the case of pneumonia referred to in Table II, severe spontaneous diarrhoea came on; and many other cases are quoted, with similar diminution of the excretions at the period of increased febrile heat, in which violent purging, sweating, or diuresis, with increase of urea and of sulphuric acid, subsequently occurred. Such discharges occurring during the progress and towards the termination of a febrile disease have been termed *critical*, the occurrence being called a *crisis*; and the particular day on which it happens, counting from the day of seizure, has been called a *critical day*. The term *crisis* or *critical* is applied because the occurrence of such discharges is usually coincident with more or less sudden fall of temperature, and general improvement in the condition of the patient, whose convalescence dates from the

critical day ; when, in common language, his disease is said to have "got the turn." In such cases, therefore, a large amount of partially metamorphosed substances are retained until they are suddenly discharged, and the system freed from the noxious compounds. Coincident with the critical discharge, the temperature is found to fall.

2. But in another class of febrile cases retention of the products of metamorphosis is not followed by such a fortunate *critical* issue. At a later period in the history of some febrile cases, with diminished excreta, it is not uncommon for *secondary inflammatory* affections to occur, as if the blood were more contaminated; and it is sometimes observed that in a patient whose excreting organs are acting copiously, there occurs a diminution of excretion when a simultaneous or subsequent development of local disease becomes manifest.

The following Table of Cases recorded by Dr. Parkes is interesting from the exactness of the observations and the coincidence of the local lesions with suppression and retention of excreta during febrile states:

TABLE III.—TABLE OF CASES OBSERVED BY DR. PARKES TO SHOW LOCAL LESIONS COINCIDENT WITH SUDDEN RETENTION OF THE EXCRETIONS IN FEVER.

Disease.	Average Temperature above 98°.	Condition of Pulmonic Function.	Condition of Cutaneous Function or Skin.	Condition of Intestinal Function.	Condition of Urinary Excretion.
Rheumatic Fever.	Fahr. 2°.		Sweating profuse.		While 400 grains more than in health were being daily excreted, <i>suddenly</i> on the eighth day a diminution of the solids took place by 602 grains; and coincident with this diminution a local lesion became developed (<i>angina faucium</i>). Next day the excretion augmented, and the local affection subsided.
Observed on the fifth, sixth, and seventh day of the disease.	Fahr. 2°.	No record.	Lessened on the eighth day.	No intestinal discharge.	
Typhoid Fever.	Fahr. 5°.		Great sweating.	Diarrhœa profuse.	While the average daily excretion for eight days was 422.848 grains, a gradual diminution continued for three days, to the daily extent of 78 grains, when pleurisy came on.
	Fahr. 5°.	No record.	Lessened much.	Diarrhœa ceased.	

TABLE III.—Continued.

Disease.	Average Temperature above 98°.	Condition of Pulmonic Function.	Condition of Cutaneous Function or Skin.	Condition of Intestinal Function.	Condition of Urinary Excretion.
Rheumatic Fever.		No record.	Sweating moderately.	Unchanged.	Considerable quantity of urine passed, containing an excess of solids. A sudden and great diminution both of the solids and fluids of the urine took place, when the joints again began to suffer, and pleurodynia supervened.

Thus it is evident, from these carefully recorded observations, "*that diminished excreta in fever are to be referred to retention of such excreta, and not to a want of formation; and that while the amount of excreta (capable of being measured) may in fact be small, the amount of tissue-change may nevertheless be great.*" The practical lesson, so often insisted on by the older physicians,—"*never to look up the excretions,*"—is thus demonstrated with scientific exactness. Another general and practical conclusion is, that the *febrile heat* cannot be measured by the amount of the excretions as a whole, nor by any ingredient of them in particular; but must be estimated in correlation with age and bodyweight. Under the same degree of heat and in the same disease different patients pass very different quantities of *urea, uric acid, sulphuric acid, phosphoric acid, cutaneous and intestinal excretions*. The same observations may be made regarding men in health. No two persons pass exactly the same amount of excretory products.

The nature of these excretory products of the febrile state teach us, however, that it is the albuminous or nitrogenous tissues of the body which are being destroyed; for the excretory products of the urine are the representatives of the azotized structures. The amount of these excretory ingredients varies considerably from day to day in fever, exactly as in health. Often there is a regular gradation of increase and decrease: the urea, for instance, may, for two or three days, slightly but regularly diminish in amount, and then suddenly augment to its highest point, again slowly to fall. The same fact may be observed with the sulphuric acid; and Dr. Parkes is led to believe that, both in health and in disease, a certain periodicity, having a range of three or five days, is connected with these gradations of increase and diminution.

The largest amount of *urea* excreted in twenty-four hours in the febrile state is recorded of a case of pyæmia, by Alfred Vogel, namely, 1235 grains. The largest amount observed by Dr. Parkes was in a case of typhoid fever, in which it amounted to 885 grains.

The largest amount of *sulphuric acid* recorded by the same observer, when no medicine was taken, was in a case of rheumatic fever. It amounted to 52.668 grains; and under the influence of liquor potassæ in the same disease, he has known this excretion rise to 70 grains—more than twice as much as in health. The largest amount of *uric acid* excreted during a febrile disease in twenty-four hours, as recorded by Drs. Parkes and Garrod, has been 17.28 grains.

“The amount of tissue destroyed in order to furnish such quantities of excreta must be enormous; and if it is recollected that little or no food is taken by the *feverish* patient, and, therefore, that no materials are supplied for the reconstruction of the textures thus melting away three times more quickly than in health, the rapid loss in weight in fever, and the impaired nutritive condition of every organ at its close, will be at once evident.”

It is not yet determined where the increased destruction of the albuminous textures takes place; that is, whether it occurs in the blood or in the organs themselves. It is only known that both the albumen and the red corpuscles of the blood are lessened in amount at the end of a febrile disease; and of the various tissues none appear to waste so fast as the muscles, and especially the involuntary ones (*e. g.*, the heart in typhus fever). The fat of the body is rapidly absorbed in fevers; and Virchow asserts that the bones also become lighter. While it is known that much of the metamorphosis of these tissues takes place in the normal way, it is also probable that there is an unhealthy or perverted metamorphosis which leads to the appearance of compounds in the excretions, either altogether foreign to the body or foreign in respect of place and time. There is evidence of this in the peculiar smell of the perspiration, in the peculiar coloring matter of the urine, as well as in the occasional excretion by it of hippuric as well as of lactic, valerianic, and other organic acids.

Next to the occurrence of preternatural heat in fever, the *excessive retention of water in the febrile system* is perhaps the most remarkable and constant phenomenon. Notwithstanding the large amount of water frequently taken to quench the extreme thirst, the quantity of the urine is lessened, and is even scantiest when the skin is driest; and the “concentration of the urine appears to be almost as good an index of the amount of fever as the temperature itself.” The excretion of water by the skin is, as a rule, diminished; and it is a well-known clinical fact that the skin is drier than usual in febrile affections. Very early in the febrile state the buccal mucous membrane also becomes sticky, and the amount of saliva diminishes; and the decrease in the quantity of the gastric fluid during fever has been proved by the well-known experiments of Beaumont on Alexis St. Martin. The intestinal juices, like the gastric, are also probably diminished, for the stimulus of food is taken away, constipation prevails, and the fæces are dry (PARKES).

This retention of water in the system cannot at present be explained; but Dr. Parkes has suggested that it may possibly be due to the presence in the blood (or tissues generally) of some interme-

diate waste product of the febrile body, of some substance which (like gelatine) has a powerful attraction for water.

Besides water, there is reason to believe that chloride of sodium is retained to a certain extent in fever, or that it passes off less readily with the urine; but much has yet to be learned of the nature of fever from investigations regarding the chemistry of the *excretions*, of the *secretions*, of the *blood*, and of the *organs*.

The Urine in Fever.—The general characters of this excretion proper to the febrile state are deficiency of water, increase of solids, if they are not retained, and especially of the *urea*, the *uric*, the *sulphuric*, the *phosphoric*, and the *hippuric acids*. The *pigments* also are increased; and the *chloride of sodium* is diminished. The deep color of febrile urine has usually been attributed to its concentration; but if febrile urine be diluted to the usual amount of fluid contained in healthy urine, it is still darker than normal urine. The coloring matter has been shown by Vogel to be increased sometimes fourfold, and it appears to contain more carbon than usual. This coloring matter in febrile urine is peculiar, and does not give any of the reactions of the bile-pigment. It may, according to Dr. Parkes, be considered as a measure of the metamorphosis of the blood-globules, which in some cases may thus be four times as rapid as in health.

Another important fact connected with the chemistry of the urine in fever is the *augmentation of its free acidity*, as measured by its neutralization with soda.

The Blood in Fever.—The most trustworthy and interesting facts connected with the chemistry of the blood in fever are,—(1.) A diminution of the alkaline salts, as shown by Becquerel and Rodier in inflammation; (2.) A diminution of alkalinity of the serum, as shown by Cohen; (3.) The diminution of the albumen after the fever has lasted for some time, with a commensurate increase in the water of the serum; (4.) A diminution in the numbers of the red corpuscles of the blood; (5.) In certain specific fevers the presence of *uric acid* has been detected; for instance, in rheumatism, by Dr. Garrod.

Of the Pulmonary Excretion, in the febrile state, little is known. Some have found the carbonic acid augmented; others have found it diminished. Dr. Wilks, of Guy's Hospital, found that the ratio of respirations to the pulse is always increased, and that the pulse may be descending while the respiration remains high. Such phenomena he considers indicative of a positive increase of function of the lungs.

It is important to determine when the blood becomes affected in fever. It has been, and still is, a favorite opinion to refer the *origin* of fever to primary disease of the blood; and in almost all specific diseases, such as in the *miasmatici*, a *fever-making cause* appears to enter the blood; at least, writes Dr. Parkes, it may be proved to enter in several cases; and a strong analogical argument can be proved of its entrance into others. The fever-making cause also reproduces itself in the blood, or in some organs; and it is now generally admitted that the first action of the febrile cause is on the blood.

The Nervous System seems to play so important a part in fever that

Virchow, in his definition, states that the essential phenomena *must* have their immediate cause in changes of the nervous system.

It is very difficult to substantiate this position, but the following general results prove the great influence of the nerves in febrile affections. Taken individually, they, no doubt, will impress different minds with different degrees of force, while collectively they cannot fail to furnish an argument in favor of the essential participation of the nervous system in fever:

1. There is the generally received physiological law, that nerves regulate the metamorphosis of tissue and the production of heat, both of which are altered in fevers (HELMHOLTZ, LUDWIG, BERNARD).

2. There are those experiments on the vagus nerve which bring about febrile phenomena, such as increased cardiac action, pulmonary congestion, anorexia, and nausea (BERNARD, PAVY).

3. There are those arguments derived from the various symptoms which announce, accompany, or terminate fever. (a.) The remarkable depression, apathy, sense of exhaustion and debility which usher in the febrile state. (b.) The shiverings, the contraction of the superficial vessels and of the skin. (c.) The increased rapidity of the heart's action, and the relaxation of the vessels, which soon follows the stage of contraction just noticed, or occurs without it. (d.) The congestion of the lungs. (e.) The periodicity of some of the phenomena of fever, and the occurrence of death or recovery on so-called critical days. (f.) The abnormal state of the secretions.

4. The fearfully rapid death which sometimes ensues in the early stage, from some unknown cause, may with justice be referred to profound nervous lesions; for there is great prostration, a galloping and early-failing pulse, and an excessively rapid respiration.

5. The effect of certain remedies, such as quinine, upon periodical febrile phenomena.

Conditions which combine to produce the Complex Phenomena of Fever.—1. The entrance into the blood of a morbid agent, and the alteration of the blood to a certain extent under its influence, come first in the order of events. Perhaps this occurs under the incubative period, when often there is no rise of temperature, no fever; that is, when no appreciable alteration of the general health can be discovered. The nature of the change in the blood is unknown.

2. When the change in the blood has reached a certain point, the nervous system, or rather that part especially connected with nutrition and organic contractility, begins to suffer changes in composition, which probably paralyze, impede, or destroy the normal molecular currents. When this occurs, the nervous symptoms of weakness, depression, rigors, contraction of some parts and vessels speedily followed by relaxation, mark the stage of invasion.

3. Various parts simultaneously, especially the muscles, and probably some of the organs, deprived in greater or less degree of nervous influence, begin rapidly to disintegrate, and by their disintegration preternatural heat is produced. Little or no fresh material is assimilated to compensate for the loss; great muscular prostration ensues; and destruction of tissue is increased by the accelerated action of the heart.

4. This metamorphosis is aided, in most cases, by the condition of the vagus and vasi motor nerves, which cause increased action of the heart and dilatation of the vessels.

5. The contamination of the blood, already produced by the morbid agent, is increased by the check which the normal extravascular currents experience, by the pouring into the blood of the rapidly disintegrating tissues, and by the continued action of the morbid agent, which in almost all cases appears to act more rapidly and more powerfully in blood rendered impure in any way, either (as shown by Dr. Carpenter) by retention of excretions, absorption of septic substances, or, as in fever, by the too rapid metamorphosis of tissue.

6. The various organs suffer (apart altogether from specific changes), and must produce increased deterioration of the blood. Thus the lungs are congested in so many cases that we can scarcely suppose proper aeration to go on; the liver would seem, from Frerich's observations, to be, in some cases at any rate, in a most abnormal condition, and to produce compounds, such as leucin, unknown in health; and the spleen in many fevers, if not in all, enlarges (in persons of a certain age), and is congested, possibly even to extravasation.

7. Food being almost withdrawn, the various alkaline and neutral salts no longer pass into the system.

8. Non-elimination of the products of tissue-metamorphoses may give rise to cerebral symptoms and local inflammation.

9. On the elimination of the fever-poison and of the products of the tissue-metamorphoses, the nerves resume their normal functions, the undue consumption of tissue is checked, and the patient regains strength and weight.

10. When coma, delirium, or stupor present themselves in the course of fever, it is the custom to refer these symptoms to the action of the fever-poison on the brain; but the cerebral functions are more probably deranged not alone by the fever-poison, which was the first and the necessary link in the pathological chain, but by the accumulation in the blood of the products of tissue-metamorphosis, and by the perverted and defective nutrition of the brain itself. Hence the symptoms in the advanced stage of many fevers are closely assimilated, although the primary poisons have been perfectly distinct (PARKES, MURCHISON).

All these events tend to render the febrile state an extremely complex one, and its investigation difficult. Our science is often spoken of as an exception to the exactness or comparative certainty of the (so-called) physical sciences; but nothing can demonstrate more clearly the claims of the Science of Medicine to exactitude and certainty than the advances made in recent years in our knowledge of the natural history of febrile diseases—their causes, their modes of propagation—their development, natural course, and termination.

Improved methods and instruments of research, and more extensive clinical instruction at our schools of medicine, have contributed to this end; and perhaps nothing demonstrates this exactness and

certainly better than the advances made in our knowledge of the "thermometry of disease," and the correlation of temperature with other morbid phenomena. By numerous careful observations it is now clearly established that the determination of the correlation of the pulse, the respiration, and the temperature of the body in disease is of the greatest practical importance, and especially when regarded in relation to the *excreta*. By such observation the natural course and termination of many diseases can be predicated with great certainty; and so our knowledge becomes more exact as to the nature and treatment of diseases. The pulse, the respirations, and the temperature, all and each of them, represent forces at work in the living body, all of which are capable of being measured with great exactness; and such measurements show how closely such expenditure of forces is related to the *excreta*, which represent the waste of our tissues in health and disease. The student or physician who continues to disregard the aid of thermometry in the diagnosis of febrile disease, or the military medical officer who ignores its value in the appreciation of feigned diseases, such as rheumatism, may be compared to the blind man guiding himself. By means of great practice and intelligence, the blind man will often proceed rightly; but the advantages of being able to see clearly are proverbially above all price. The necessity of using the thermometer will also soon become known to the general public, and patients will become dissatisfied if all known means of investigation are not employed in appreciating the nature of their malady. For many years the German student and physician has been familiar with its use; but, with the exception of Dr. Parkes, and the pupils he taught when clinical professor in University College Hospital, the usefulness of the thermometer in recognizing febrile diseases does not seem to have been hitherto sufficiently appreciated in the medical schools of this country.

SECTION II.—INFLAMMATION.

Definition.—*A complex morbid process characterized,—(1.) By a suspension of the concurrent exercise of function among the minute elements of the tissue involved; (2.) By stagnation of the blood and abnormal adhesiveness of the blood-discs in the capillary vessels contiguous to the tissue-elements whose functions are suspended; (3.) By contraction of the minute arteries leading to the capillaries of the affected part, with subsequent dilatation and paralysis of the contractile tissue of the affected bloodvessels. The nutritive changes between the blood and the minute component elements of the affected tissue become visibly altered, and although an appreciable interstitial exudation does not necessarily follow, yet a constant tendency betrays itself to the occurrence of an interstitial exudation, but which, under proper regimen and proper remedies, is often abortive. When an exudation follows as a result of the inflammatory state, it is apt to be associated with an unhealthy condition of the blood, and of the blood-plasma, and to be associated with varied forms of new growth, according to,—(1.) The elementary structure in which it*

occurs ; (2.) *The special zymotic, constitutional, or local disease with which this complex morbid process may coexist ; and (3.) According to the progress of the inflammation, the amount and suddenness of the effusion, the extent of tissue involved, the diminished vascularity, and the powers of absorption of the surrounding parts.*

Pathology.—As it is not possible clearly to define the limits of natural processes, it is not possible to give a correct definition of inflammation. It is a process the most important of all morbid states ; and a knowledge of its phenomena, the laws which regulate its course, and the relations which its several events bear to each other, have been always considered as “the keystone to medical and surgical science,” and the “pivot upon which the medical philosophy of the time has revolved.”

It is not wonderful, therefore, that much has been written on this subject, more especially since microscopic research has been brought to aid in the investigation. Among the many who have investigated this morbid process with success, and by whose original observations its study may be said to have begun, the names of Wilson Philips, John Thomson, Gendrin, Kaltenbrunner, Gerber, and Müller ; and more recently those of Alison, Lebert, Gulliver, Addison, C. J. B. Williams, Bennett, Wharton Jones, Henle, Virchow, Paget, John Simon, and Joseph Lister, are well known ; and no account of inflammation can be complete which does not embrace the results of the labors of these men.

The early experiments which illustrate the nature and phenomena of inflammation, have been made chiefly on the web of the frog's foot, as well as on the folds of the frog's mesentery ; and the phenomena are found to correspond in all essential points, with the results of experiments performed on the more or less transparent parts of warm-blooded animals, such, for example, as the wings and ears of bats, the ears of rabbits, the mesenteries of these animals, and the brains of rabbits and of pigeons. As a general result of such experiments and observations, it may be stated, that the chief constituents of the inflammation-process, are to be found in altered conditions of the healthy nutritive changes—the phenomena of the abnormal state becoming more or less obvious by the redness, swelling, heat, pain, impairment of function on a large scale, and sometimes exudation in the part affected.

Phenomena and Theory of the Inflammatory Process.—The process is one in which many stages of morbid action are passed through, and which reaches its acmé when the serum of the blood and the *liquor sanguinis* transude through the walls of the bloodvessels of the inflamed part, without rupture, into the surrounding texture. This has been termed “exudation.”

The series of complex changes through which the inflammatory process is seen to proceed, as observed in the transparent parts of animals under the microscope, are found to occur nearly in the following order: 1st. The beautiful experiments and observations of Mr. Joseph Lister, Professor of Surgery in the University of Glasgow, clearly prove that a suspension of the concurrent exercise of function among the minute elements of the tissue involved, is the

primary lesion in the congestion of inflammation, and which immediately leads to—2d. Inflammatory derangement of the blood, which, in the vicinity of the impaired tissue-elements, tends to assume the same characters as blood always assumes when it is in contact with ordinary solid matter, and which renders it unfit for transmission through the bloodvessels. But a return of the tissue-elements to their usually active state, will be associated with a restoration of the blood to the healthy characters which adapt it for circulation (*Royal Society, June 18, 1857*). 3d. The arteries of the affected part are narrowed, and the blood flows through them with greater rapidity. 4th. The same vessels subsequently become enlarged, and the current of blood is slower, although uniform. 5th. The flow of blood becomes irregular. 6th. All motion of the blood ultimately ceases, and complete stagnation ensues. 7th, and lastly, The liquor sanguinis may be exuded through the walls of the bloodvessels, sometimes accompanied by the extravasation of blood-corpuscles, owing to rupture of the capillaries.

These different phenomena are associated with the production of the more obvious symptoms, namely, redness, pain, heat, and swelling. But although these changes are here mentioned consecutively, it is not to be understood that in every instance of inflammation, such changes can be traced in distinct succession. The changes are to be studied as *nearly concurrent*, rather than as a distinct series of events, of which each stands in the relation of a consequent to one or more of its antecedents; so that, starting from impaired function of the element of tissue, the stagnation of blood in the capillary vessels, we must observe the various stages in the process almost as concurrent phenomena, which, for the purposes of study, are here enumerated in sequence.

An analysis of these concurrent phenomena has shown that the conditions for the healthy nutrition of the part are materially changed, being somewhat as follows:

I. The supply of blood to the part is altered,—(1.) By the changes in the bloodvessels, especially the narrowing of the arteries, and subsequent enlargement of the capillaries; (2.) By the mode in which the blood moves through them.

The narrowing of the arteries, in the first instance, may be demonstrated under the microscope, by the application of warm water simply to the web of the frog's foot; and the same phenomena are presumed to occur in man, for the following reasons: Sudden operations of the mind, and the application of cold produce paleness of the skin—an effect which can only arise from contraction of the minute arteries, and the diminution of the quantity of blood thereby conveyed by them. The subsequent enlargement of the capillaries is presumed to be a constant event in the inflammation of a part. It usually extends to some distance around what may be considered as the chief seat, centre, or focus of diseased action, and in some textures the enlargement and reddening are confined to the vascular parts in the vicinity. To this condition of the blood and bloodvessels, is to be ascribed the usually first observable symptom of inflammation in a part, namely, the redness. But there are also

many circumstances under which inflammation has existed, and yet no redness is apparent in the part itself. Thus we often open the body of a patient that has died of phthisis, and find the intestine ulcerated; but, so far from being redder, it is paler than natural, and, so far from being thickened, it is thinner than usual. We often find the cartilages of the joints ulcerated, and yet not a trace of a red vessel. In cases of bronchitis, with purulent expectoration, if the lungs be washed, so as to remove the morbid product, the most experienced anatomist may be unable to determine whether the parts are in a state of health or disease. Take the arterial system, and how often do we find the aorta thickened and thinned, softened and indurated, ulcerated, and its elasticity entirely destroyed, and yet not a red vessel to be seen; and when the patient has neither complained of the slightest sensation of pain, nor of any feeling of heat in the part during life? A large abscess may form in the brain or areolar tissue, or pus may be effused into the cavity of the abdomen, without any appearance of redness, or even evidence of having been preceded by any suffering. Although in certain parts, as the cornea and the articular cartilages, the ulcerated intestine, or the bronchi, the arterial tissues, and the seats of abscesses, the previous existence of inflammatory action is obvious from the effects produced, and where no bloodvessels existed, obvious to the eye, assisted or not by the microscope, yet it is, for the most part, found that enlargement of the bloodvessels of the adjacent parts, and especially of those from which the diseased part derives its nutrient supply, is a constant phenomenon, purely functional, and which appears to be developed indirectly through the medium of the nervous system. In inflammation of the cornea, for instance, the bloodvessels of the sclerotic and conjunctivæ are enlarged. In ulceration of the articular cartilages, the surrounding synovial membrane, and the articular extremities of the bones, are more fully pervaded with enlarged bloodvessels. The *vasa vasorum* of the aorta, round the morbidly thickened part, are also the subject of enlargement, and the channel of increased supply of blood. There is, therefore, no doubt that the conditions favorable to the existence of redness are always present to a greater or less degree at the early period of inflammation; and whether the redness be always present, or only slightly perceptible, the same impairment of function among the minute elements of the tissue, and increased adhesiveness of the blood-discs, take part in the development of the inflammatory process.

The enlargement of the bloodvessels varies. It may be hardly perceptible, or it may increase their diameter to two or three times their natural size. John Hunter established this stage of the inflammatory process in the ear of a rabbit, by thawing it after it had been frozen: the rabbit was killed during the process, and the head being injected, the two ears were removed and dried. Wood-cuts representing the comparative conditions of the two ears may be seen in the first volume of Paget's *Surgical Pathology*, page 295. The bloodvessels of the inflamed ear became greatly larger than those of the healthy one, and it was found that arteries before invisible, in the

healthy state of the rabbit's ear, were brought clearly into view during the stage of the inflammatory process.

The redness of an inflamed part is of various intensity and shade, according to the degree of the inflammation, its stage, and the structure of the part affected. Its shades pass from a light rose-color to a deep crimson, or even purple. It assumes the form of points where congeries of minute bloodvessels are concerned; or streaks, as where the vessels of fibrous structures are inflamed, as in tendon; or a series of minute and fine ramifications, as in synovial structures; and generally it may be stated that the form of the redness derives its character from the normal arrangement of the capillaries of the part. The redness is most intense towards the centre of diseased action, gradually softening down towards the circumference, where the conditions of health exist. This gradual shading off serves to distinguish the redness of inflammation from the redness of extravasation. The margin of an extravasation is defined, its redness cannot be removed by pressure; while the disappearance of inflammatory redness under pressure is, to a certain extent, a measure of the activity of the circulation in the part. The brighter hues generally attend ordinary active inflammation;—the darker hues of inflammatory action are generally associated with some specific cause of disease, a feeble action of the parts, or a tendency to gangrene. The increased depth of color is mainly due, in the first instance, to the congestion and stagnation of blood in the existing vessels, and not in any measure to the formation of new ones. The redness, however, always appears more than proportionate to the enlargement of the bloodvessels; and we find that the red corpuscles are intensely adherent in the enlarged capillaries.

The dilated vessels of an inflamed part appear crammed with red corpuscles, which lie or move as if no fluid intervened between them, or as if they were imbedded in a hyaline substance due to the solidification of the fibrine of the liquor sanguinis. An increase of redness is sometimes seen to depend upon extravasation of blood, or the effusion of the coloring matter of the blood-corpuscles, as well into the spaces between the blood-corpuscles as into the adjacent tissue through the walls of the bloodvessels. Lastly, the redness is sometimes intensified (as HUNTER first suggested, and microscopic examination subsequently proved) by the passage of the blood, unchanged, from the arteries into the veins. No new formation of bloodvessels is necessarily concerned in the redness of inflamed parts. It is only when inflammation has subsided that new vessels are formed, and pass into any new growth of tissue which may have arisen, as if for its nutrition, development, and continued growth, or to effect its subsequent removal, degeneration, decay, or absorption.

Peculiar changes of shape are associated with enlargement of the bloodvessels, consisting chiefly of tortuosity of distribution and aneurismal or varicose dilatation. The aneurismal or varicose state is seen to take place most frequently in the soft textures, as in the brain, where it is a frequent condition of the inflammatory red softening (KÖLLIKER and HASSE); and in subcutaneous tissue, the points

of what appears to be extravasated blood are aneurismal dilatations of capillary vessels filled with the red corpuscles (LEBERT).

These varied conditions of the bloodvessels affect the motion of the fluid in the part, and consequently the supply of blood for the purposes of nutrition. Generally it may be stated that there is stagnation of the blood in the focus or centre of severe inflammation; and this stagnation is surrounded by a state of fulness of vessels and slow movement of the blood, while around, and more distant still, there is fulness of the vessels, with a rapid movement of the blood. From the discrepancy existing among observers regarding the statement as to whether the motion of the blood is slower or quicker when the vessels are contracted or dilated, there is evidence that the contraction alone of a vessel, or its dilatation alone, is not always sufficient to cause the current of blood to be either slow or quick. Other conditions are at work which contribute in no small degree to accelerate or slow the rate of movement in the vessels. Besides the force of the heart's action, there is a mutual relation which subsists between the blood and bloodvessels and surrounding tissue, which materially influences the motion of the blood. In the healthy body this mutual functional relation between the minute elements of tissue and the blood is necessary to maintain it in a state fit for transmission through the vessels. The mere contraction of the arteries leading to a part does not tend to stagnation of the blood in the capillaries of the inflamed part; on the contrary, the movement onwards of the blood in the vessels is influenced or modified by the vital functional processes going on between the capillary vessels and the surrounding elements of tissue, and which has been variously named the "capillary force," the "vital force," the "nutritive force;" it is also mainly influenced by the action of the heart itself, and by the physical condition of the vascular tubes through which it has to pass. Accordingly, at first, with contraction of vessels, the current has been described as being quickened. It also sometimes slackens, or even retrogrades for a time, and not unfrequently oscillatory movements may be noticed. But when dilatation is complete, the blood flows with rapidity, and a greater quantity passes during a given time than in the unexcited state of the parts. This is known as the state of "determination of blood to a part," or "active congestion." The natural function of the part thus becomes simply exalted; and it may be said that a step beyond this will pass the confines of that neutral ground which exists between health and disease. With an increased circulation, and such "determination of blood to a part," functional activity is not only maintained, but is promoted and increased; and unusual transudation of the nutrient material may take place, chiefly of the serum of the blood. Hence the œdema which surrounds an inflamed part. After a time the motion of the blood becomes slower, while the volume propelled is increased, and the retardation gradually increases till the blood-corpuscles are no longer propelled, floating in their *liquor sanguinis*; but accumulating in masses, they advance by a jerking intermittent motion, till at last complete stagnation takes place. The blood-corpuscles now

detained exhibit a marked tendency to adhere alike to the walls of the vessels and to each other; thus accumulating together and sticking in the capillaries, while the *liquor sanguinis* flows onwards. To this condition the term "stasis" has been applied. In the immediate neighborhood, and surrounding the part which is in the condition of *stasis*, the circulation of the blood goes on with increased rapidity: it may even pulsate in the arteries and oscillate in the veins, while it moves with a uniform but rapid flow through highly distended but less turgid vessels. When these conditions exist simultaneously, and the true morbid process is completely established, the capillary vessels may burst, causing hemorrhage or extravasation into the surrounding tissue, or the serum and *liquor sanguinis* may transude through their walls, without rupture, into the surrounding texture.

The "determination of blood to a part" here noticed, characterized by dilatation of the arteries with increased flow of blood through the capillaries, must be distinguished from the "congestion of inflammation," characterized by the accumulation and stagnation of red and white corpuscles in the vessels, tending to be abnormally adherent to each other and to the vessels. Both of these phenomena, namely, "determination" and "congestion," may result from irritation. The dilatation of the arteries seems to be immediately developed through the medium of the nervous system, while the accumulation of the blood-discs and stagnation of the blood is the immediate and direct result of impaired or suspended function of the minute tissue-elements contiguous to the capillary vessels.

The "determination of blood" and dilatation of the arteries lead to no change in the quality of the blood itself; on the other hand, accumulation and stagnation of blood, in the congestion of an inflamed part, are associated with increased adhesiveness of the red and white discs. Mere determination of blood becomes obliterated after death by the post-mortem contraction of the arteries, whereas the congestion of inflammation is persistent. It is an evidence of organic lesion declaring itself as distinctly in the dead as in the living; and thus the most important, if not the only sign of the early stage of inflammation having occurred during life is recognizable, on dissection, by the intense redness due to the accumulation of red discs adherent to each other in the minutest ramifications of the vessels, and not due to distension of the vessels merely.

Such is a statement of the facts ascertained regarding the early phenomena of the inflammatory process; and they are of such a kind that, with the facilities of study which ought now to be within the reach of every student of medicine, he ought to make such experiments as have been already noticed, or see them made by others, and thus really appreciate the steps of that morbid process which he requires to treat so extensively in practice, and of which he can form but a faint conception from the most lucid description.

II. The constitution of the blood is altered as regards its adaptability to nourish the part.

The nature of this alteration cannot be chemically expressed; but microscopical observation has established a fundamental fact, namely,

that the tissues through which the blood flows have such special relations to the living fluid that, in the healthy state, the functional activity of the minute tissue-elements maintains the blood in a state fit for transmission through the bloodvessels; and the first change observed in the blood, subsequent to any impairment of function of tissue-elements, is an increase of adhesiveness of the red as well as of the white corpuscles: but the white corpuscles are now known to be susceptible of much greater adhesiveness than the red; so that slight irritation, leading to impairment of function, causes stagnation of the white sooner than of the red discs. The blood is not thus altered in the first instance throughout its whole mass; but the change is a local one, confined to the seat of the inflammatory process. At one time it was believed that the blood was altered in its constitution chiefly by an increase of the fibrine and the white corpuscles;* but it is now found that the white or rudimental corpuscles of the blood cannot be separated from the fibrine by any known process; consequently the relative amount of fibrine cannot be correctly stated in relation to the blood. And, as in many inflammations these corpuscles are increased, as well as in many conditions, such as pregnancy, in which no inflammatory process exists, the blood is similarly altered, it is not known how much of change is due to fibrine or how much to the white corpuscles. The generation and accumulation of large numbers of white corpuscles in the vessels of an inflamed part is not now received as a fact. The phenomenon may be true as regards some frogs, but not as regards warm-blooded animals; and it is consistent with the experience of three most eminent pathologists who have experimentally examined this subject—namely, Mr. Wharton Jones, Dr. Hughes Bennett, and Mr. Paget—that an especial abundance of white corpuscles in the vessels of an inflamed part is neither a constant nor even a frequent occurrence. Dr. Hughes Bennett's researches, relative to leucocythæmia, have shown that even the most extreme abundance of white corpuscles in the blood has no tendency either to produce or to aggravate inflammations.

A remarkable phenomenon presented by the red blood-corpuscles in inflammation was first observed in 1827 by Mr. Lister, Sr., and by Dr. Hodgkin, and afterwards accurately described by Mr. Wharton Jones.

They observed that when healthy blood is received on a glass plate, or the clean surface of a polished lancet, and immediately examined, the corpuscles lie diffused in the *liquor sanguinis*, but in about half a minute they run together into piles or rouleaux, which arrange themselves in small meshed networks. But if a drop of blood from a patient with acute rheumatism, or with an inflammation, be similarly examined, piles of red corpuscles instantly form,

* Andral and Gavarret showed that the proportion of fibrine in the blood was augmented in inflammations, when sufficiently severe or extensive to affect the system. In health the average proportion is three parts in 1000, and in cases of severe inflammations it has been found to rise as high as eight, nine, or ten parts in 1000. This increase commences as soon as the inflammation is established, and ceases when the process begins to decline.

and are clustered into masses, leaving a network with wide interspaces.

This appearance of itself, however, is not a sure sign of inflammation. It may be observed in the blood of the chlorotic female as well as in the pregnant one; in those also in whom a plethoric condition as regards the blood exists; in persons in health, whose circulation has been much accelerated, as by violent exercise; and it appears to be the natural state of the blood of horses. It is a phenomenon resulting from an increased tendency to aggregation of the blood-corpuscles, and gives a granular appearance to a thin layer of blood when viewed with the naked eye. When blood is drawn off in quantity, the phenomenon is associated with the formation of what is termed the "buffy coat," as the clustered blood-corpuscles, rapidly sinking, subside to some distance below the surface before the fibrine and the white corpuscles begin to coagulate.

However indefinite and uncertain the changes may be, as observed upon a small portion of the blood, it cannot be doubted that the blood stagnant or retarded in an inflamed part undergoes important alterations; and by a constant succession of such changes the whole fluid may come at length to be materially altered, as indicated by the general effects and constitutional disturbance, extending throughout the nervous and the vascular system, and which may ensue in the train of an inflammation of purely local origin. It is probable that local changes ensue in the blood similar to those we shall have to notice as taking place in the products of growth in and amongst the elements of tissue during the inflammatory process. There is no doubt, as Wharton Jones has shown, that fibrinous coagula occasionally form, and even degenerate, within the bloodvessels. When the stagnation of the blood is not constant, these fibrinous coagula are carried away into the general circulation, giving rise to the phenomena of embolism (to be afterwards described) in the capillary vessels of some of the more solid viscera, such as the brain, lungs, liver, spleen, or kidneys. By the degeneration of such coagula the whole mass of blood may be infected, and constitutional disturbance excited, producing sometimes various and wide-spreading suppuration,—as when purulent infection is consequent on local injury, or when a blood-clot passes upwards, and becoming lodged in the cerebral vessels, induces the state known as softening of the brain.

There are many points or questions deserving of attention regarding the theory of the inflammatory process; but it is also obvious that in a handbook such as this, any mere analysis of speculative doctrines ought not to take up much space. The following statement will therefore merely embrace as much as possible of those topics of special interest which a more extended and accurate physiological knowledge of the process of inflammation has shown to be the proper objects of more extended inquiry.

In the first place, as to the primary seat of the inflammatory process, there can be little doubt, from the phenomena already described, as well as from the results of dissection, which show the progress and effects of the process, and from the experimental

researches of Hunter, Thomson, Wilson Phillips, Hughes Bennett, Wharton Jones, John Simon, Paget, Lister, and other observers, that the vital morbid process known as "inflammation" is connected with the minute capillaries and the most minute elements of tissues which they nourish. Questions relative to the theory of the process are therefore found to be intimately connected with the histological and physiological relations of these parts.

During the earliest period of the process—the period of increment, or of incubation, as it has been termed—it appears to be the inherent properties of the minute component elements of tissues which first undergo a change, and, combined with the reflex actions of the nervous system, seem to maintain, to promote, or to increase the activity of the subsequent stages.

The simplest effects upon the minute elements of tissue, and upon the bloodvessels, are seen to follow the application of the mildest or slightest physical or chemical agents, but which, operating powerfully, are also capable of extinguishing altogether the life of these elements of tissue. When the action induced is mild and gentle, the tissues become incapable of performing their wonted functions; and, provided the mechanical or chemical agency has not been too severe, the impairment of function may subside, and the tissues will return to their normal state of functional activity. This is "resolution" of the inflammation.

Such irritant causes acting either immediately from without, or through the blood, or through the instrumentality of the nerves, each component texture of the part becomes affected as soon as it is brought in contact with the irritant. A gradual contraction of the arteries takes place—the contraction following at some interval after the application of the stimulus—is slowly accomplished, and persists for a variable length of time. Relaxation then no less gradually ensues, when the capillaries open up and slowly dilate till they acquire a size larger than they had previous to the application of the stimulus.

The minute arteries have been shown by the histologist to possess in abundance the structural elements of the non-striated contractile tissue; and in this respect they closely resemble the constitution of the muscular fibre of the intestine. Accordingly, the contractions they undergo have been considered as analogous to *spasms* (as Cullen first suggested); while the succeeding dilatation may be of the nature of *relaxation*, and ultimately of *paralysis*. This paralyzed state is shown from the fact that the same vessels now dilated will not contract upon a re-application of the same stimulus which before made them contract. If the stimulus is made with a needle upon the vessels in the transparent parts of an animal, the needle may be repeatedly drawn over such dilated vessels and no contraction will follow; but with a stronger stimulus, such as that of heat, they may be made to contract again, and even close; and this state of contraction may persist for a whole day, before the vessels again open up and permit the blood to flow (PAGET). On the other hand, the true capillaries seem totally destitute of any structure known to be contractile. They merely consist of a delicate homogeneous

membrane, beset with occasional nuclei. A film of collodion is not more homogeneous nor more continuous than the membrane of a capillary (VIRCHOW). Whereas the minute arteries (some of them less even in calibre than capillaries) possess distinct coats, one of them consisting of a single layer of muscular (or contractile) fibre-cells, wound spirally round the internal membrane of the blood-vessel, so as to encircle it from one and a half to two and a half times. The arteries, to their smallest branches, are sometimes contracted to absolute closure, and at other times are widely dilated; whereas the capillaries are never entirely closed, nor do they present any variations in diameter which are not due to the elasticity of their parietes (LISTER, l. c.).

The most interesting point in the whole process is perhaps that which embraces an inquiry into the cause of the "stasis," or stoppage of the blood, and the exudation of the *liquor sanguinis*, which are the most difficult phenomena to explain consistently with physiology. This is a point which I think the observations of Professor Lister have so very beautifully illustrated; but the explanations of other eminent pathologists and experimentalists, if not universally satisfactory, serve to present the subject in a variety of aspects to the mind, which cannot fail to be both interesting and practically instructive. Henle, Simon, Bennett, Williams, Rokitsansky, and Paget, have all helped to elucidate the process by the following theories:

The theory of Henle, or, as it is sometimes called, the "neuropathological theory," assumes that the stimulus, acting on the sensory nerves of the part, excites in them a state which, being communicated to the spinal nervous centre, is reflected on the vascular nerves, occasions their paralysis, and therewith paralysis also of the contractile coat of the bloodvessels. Various modifications have been made upon this theory; but as the phenomena have been seen to take place in the case of absence of a spinal cord, and in division of the roots of the nerves, and in section of the lumbar and sciatic nerves, such facts are subversive of the hypothesis. Henle considers the stasis as a necessary physical consequence of this dilatation of the bloodvessels, and this stasis, together with the relaxed and dilated state of the vessels, favors the exudation of serum, the consequence of which is, that the plasma of the blood in the part becomes inspissated by a preponderance of albuminoid matter over the salts. This inspissation of the plasma determines endosmotic changes in the red corpuscles, in consequence of which they are disposed to aggregate.

Simon propounds the view that the phenomena are due, not to a reflex action, but to a direct change effected by the living molecular structure of the part on the blood which traverses it, or on the vessels which convey that blood.

Bennett ascribes the change as due to a vital force actively operating through the tissues which lie outside the vessels, and which is the only active agency causing the approach of the colored particles to the capillary walls of the bloodvessels, and the passage through them of exudation.

Paget supposes a mutual relation to exist between the blood, its vessels, and the parts around, which being natural, permits the most easy transit of the blood, but being disturbed, increases the hindrances to its passage.

Dr. C. J. B. Williams considers that an essential part of inflammation is the production of numerous white globules in the inflamed vessels, and that the obstruction of these vessels is mainly due to the adhesive properties of these globules.

Rokitansky is of opinion that the condition of *stasis* proceeds,—1st. From the sticking together of the blood-corpuscles, the heaping up and wedging of them in the capillaries, while the plasma in part flows off towards the veins; 2d. From the inspissation of the plasma, occasioned by the exudation of serum through the dilated and attenuated walls of the vessels, and its saturation with fibrine and albumen; 3d. From the heaping up of the colorless corpuscles, *i. e.*, the nucleus and cell-formations, together with blood-globules; from their sticking together, and from the delicate hyaline, fibrinous coagula which develop themselves among them. Rokitansky considers this to be the most important moment in the inflammatory process, since on the one hand it very specially throws light upon the phenomena of *stasis*, and on the other hand it comprehends the plastic processes which take place in the heaped-up and stagnant blood. It separates in this way the process of inflammation from a merely simple one of exudation. The elementary formations above-mentioned are not merely swept together towards the place of *stasis*, but they originate as new formations in the stagnant blood, which generally presents remarkable alterations.

Wharton Jones describes the progress of *stasis* as consisting,—1st. Of the adhesion of collapsed and dark-red blood-corpuscles to the walls of the vessels; and 2d. The adhesion of other blood-cells to these. The first adhesion of the blood-cells usually takes place at a bifurcation, and the stagnation of blood is seen to begin in those capillaries which are least in the direct course from the artery to the vein, depending in a great measure upon the inspissation of the plasma, or its increased quantity of fibrine and albumen.

Whatever explanation may be given or accepted as to how the phenomena of inflammation in a part are brought about, our views regarding the essential nature of the process have been hitherto modified according as this complex morbid state has been studied by its effects as seen on the dead rather than on the living body. There are some especially eminent pathologists whose combined observations have of late done much to convey a clear notion of the essential nature of this complex process, namely, Alison, Virchow, Bennett, Simon, Goodsir, Redfern, and Lister. While Dr. Bennett regards an exudation from the bloodvessels as the necessary constituent of inflammation, Alison and Virchow, on the other hand, recognize the morphological changes of the living tissues, such as have been described in inflammation, as betraying merely a *tendency* in a part to such a local change as exudation amongst its structure. That *local tendency* may be so slight that hardly any difference can be appreciated between the healthy changes attendant on normal

nutrition, and those changes between the blood and the minute tissues which are of such a kind that a morbid change (inflammation) is established in the elementary components of the tissues themselves, without any appreciable exudation having taken place either amongst the interstices or upon the free surfaces of membranes. To such a condition Virchow gives the name of *parenchymatous inflammation*, meaning thereby that it is a process established locally between the capillaries, the blood, and the component elements of tissue, and expressed by a tendency merely to the effusion from the bloodvessels of such plastic material as may eventually take place.

Inflammation may thus exist as a local morbid process, characterized by an abnormal condition of the nutritive changes between the capillaries, the blood, and the component elements of a texture, without any appreciable exudation. Such an abnormal condition will, under proper regimen and proper remedies, in a case of simple inflammation, seen from the first, completely subside, no interstitial exudation ever taking place.

Examples of this simple form of inflammation have been fully illustrated by Goodsir and Redfern in this country, by their demonstrations of what takes place within the large cells of cartilage. The cells become larger, the number of nuclei increases, and some, or all of them, may undergo fatty metamorphosis under the influence of this the simplest form of inflammation, and which is only manifested by this abnormal nutritive process between the blood and the cells, and which at once leads to these changes *within* the cell-elements of tissue, described by Virchow as a "cloudy swelling" of these parts, *e.g.*, the cells of the uriniferous tubes, and those of the mucous membrane in the state of catarrh. In this abnormal nutritive process, however, there is a constant tendency to the interstitial exudation of a hyaline material, which may become fibrous or filamentous, and ultimately soft and gelatinous. Virchow, Weber of Bonn, and His, have demonstrated similar changes in the cells of the cornea.

Thus the minute and penetrating observations of Virchow have given a more comprehensive meaning to the process of exudation than it has hitherto, in this country, been understood to signify; and such alterations as he and others have described in the elements of the tissues of an inflamed part have been in a great measure overlooked, except by Dr. Alison and Mr. Simon. The latter especially states that the irritation of the inflammatory process is independent of the nervous influence, *but is a direct change operated by the living molecular structure of the part on the blood which traverses it, or on the vessels which convey that blood.* Dr. Alison, also, long recognized the *tendency* to interstitial exudation as attending such vital changes in the constituent elements of a part, and which entitled it to be considered inflamed. The accurate observations of Virchow, Goodsir, and Redfern have shown that such primitive changes do take place before those more palpable phenomena occur which constitute the exudation as described by Bennett, namely, the exudation of de-colorized lymph into the interstices between the constituent elements

of a texture. Both sets of phenomena alike show that *inflammation is only one of the various shades of deviation from the normal process of nutrition,—a diseased action tending to a local lesion* (*British and Foreign Medico-Chirurgical Review*, January, 1854). That the irritation of inflammation is in some measure independent of the nerves, the following interesting experiment, related and performed by Mr. Simon, may be quoted in proof:

“A patient had complete anæsthesia of the fifth nerve, dependent (as a post-mortem examination subsequently showed) on its organic disease; the conjunctiva, as well as the integument of the face, was utterly insensible: not only was the function of the nerve destroyed, but those reflexive nutritive changes of which I have already spoken had taken place, and had exhausted themselves; showing that the nerve was spoiled for participation in the acts of nutrition (whatever they may be) no less than for its more obvious uses as a medium of conscious sensation; the cornea had undergone ulceration, and had healed again. The following experiment was carefully made: The lids being held open, a single granule of cayenne pepper was laid upon the insensible conjunctiva; in a few moments it had become the centre of a very distinct circle of increased vascularity, the redness of which slowly became more and more distinct as long as the stimulus was suffered to remain, so that, in its removal, there was a very evident circumscribed erythema on the surface of the membrane. I considered myself justified in believing that this change occurred without any intermediate nervous excitement; not only because the history of the case would lead me to consider the fifth as annihilated; not only because the experiment was totally unattended with sensation; but likewise because there was the very remarkable absence of that sympathetic phenomenon which the faintest remnant of nervous excitability would have produced—namely, there was not the slightest trace of lachrymation” (*Lectures on General Pathology*, p. 76).

Further evidence might be submitted from the papers of Mr. Joseph Lister to the Royal Society, already referred to.

Such being the essential nature of inflammation, it is easy to understand how reasonable is that doctrine which teaches “that the process of inflammation is susceptible, at all times and in all countries, of very great variety as to extent or intensity, and especially as to the constitutional affection associated with it or consequent upon it.”

Products, Effects, or Events of Inflammation.—Care must be taken not to put the *products* of inflammation in place of the *symptoms* of inflammation. When the local impairment of function of the minute elements of tissue in process of inflammation *is confined to a small space, or is carried on upon a minute scale, or rapidly abates*, the inflammation is said to *terminate by resolution* as a general principle; that is, the abnormal action ceases, interstitial exudation does not take place, the tendency to further impairment of function is subdued and passes off, and the part is left apparently as it was before. If, however, interstitial exudation has taken place, and *resolution* is to be effected, the return of the part to health may be followed, for some time, by some impairment of its structure and function.

After the process has thus gone a certain length, an increased local growth of cells, and their liquefaction or reduction to a state capable

of absorption (what Dr. Addison calls *cell-therapeutics*), are essential to the restoration of the part. Before the process has attained such a length, however, *resolution* may be simply effected by a gradual return of all the parts to a natural state: a mere retracing of the steps by which the natural actions had been departed from sufficiently describes the process (PAGET).

The process of *resolution* has been closely watched by Mr. Paget. He has seen, in those cases where impairment of function and actual lesion had taken place, that fragments of fibrine, washed from the blood in the vessels of the injured parts, were borne along and floated in distant vessels. The observations of Dr. Kirkes, also, leave no doubt that similar changes may occur in warm-blooded animals, and may be the source of great evils; may be, indeed, productive of some of those constitutional effects yet to be noticed, by carrying the materials of diseased or degenerate blood from a diseased organ to one that was previously healthy. When the disappearance of the inflammation is unusually sudden and rapid, the event is technically called "*delitescence*," and if at the same time the symptoms of inflammation appear at another part not anatomically connected with the part first diseased, the event is called a "*metastasis*."

When the process does not confine itself to the simple expression of altered nutritive changes between the constituent tissues of a part and the blood; but when the tendency to exudation amongst the interstices of texture continues, and does not subside, as already explained,—namely, by *resolution*,—then it is that (1) such a material is separated from the blood as will become a medium or *nidus-substance*, in which many changes connected with the growth of new particles, granules, or cell-forms will take place, and the phenomena of which have been so well described by Bennett, Gluge, Paget, Virchow, Beale, and John Simon; and (2) coincident with this exudation, and the changes which it undergoes, the tissue of the part itself sustains serious alterations. For in all such inflammations especially of the more vascular parts, when there is increased exudation from the bloodvessels, there is a great deterioration of the surrounding elements of tissue. The texture is rendered soft and easily torn, and by such changes of cohesion the elasticity of parts (a circumstance often of very primary importance) becomes greatly altered and impaired.

These changes, therefore, Mr. Paget happily describes as consisting of,—(1.) *Productive* effects—that is, effects resulting from the growth of new particles, granules, or cell-forms, from pre-existing germinal elements of tissue, and which are susceptible of further development, and also of *degeneration*; (2.) *Destructive* effects, such as *softening*, *degeneration*, *absorption*, *ulceration*, and *death* of tissue.

Productive Effects of Inflammation—Inflammatory Effusions or Exudations.—These consist of,—1. Serum; 2. Blood; 3. Fibrine; and 4. Mucin. These last two are the only *true inflammatory exudations*.

1. *Serous Effusions*.—The effusion of pure serum is said to be very rare. In inflammation of a serous membrane, as the pleura, the fluid effused is not only greater in quantity than natural, but it is also greatly altered in quality. In health the serous secretions

are little more than pure aqueous vapor, with a trifling addition of saline matters; but when they occur in an inflamed part, they contain a considerable quantity of albumen, sometimes a portion of fibrine, and at other times the secretion appears to be the pure *liquor sanguinis* which is effused, entirely unchanged in its physical properties. The quantity effused varies, according to the part affected, from perhaps a portion of an ounce to a few pints, or even more than a gallon.

This product of inflammation, but mixed with fibrine, may be seen in the fluid contained in blisters raised by counter-irritants in a healthy person; also in the fluid of *peritonitis*; of *pleurisy* and of *pericarditis*: such also is the fluid that fills the early vesicles of *herpes* and *eczema*, and other cutaneous eruptions. It is also seen in the fluid which surrounds an acute, deep inflammation beneath the skin. The fluid of a common *hydrocele* is another example of serous effusion. The phenomena associated with the production of such a secretion, may be often seen surrounding a phlegmon, or boil of large size. While the centre, or *core* of the boil, is hard, it is surrounded by textures, into which the *effusion* that has taken place, is *serous*. Such serum is to be seen to a great extent in *pelvic cellulitis* in its first stage. A very demonstrative and interesting example of this is given by Professor Simpson, of Edinburgh, in the *Medical Times and Gazette*, for 1859, p. 27, July 9. In such cases the fluid fills the areolar tissue which immediately surrounds the inflamed parts; and when the finger is pressed firmly on the part, the fluid is displaced into the adjoining areolar spaces, which yield to receive it. When the finger is withdrawn, the fluid does not immediately return, but an impression is left in the shape of a pit. The part which is the seat of *serous* effusion is then said to *pit on pressure*, or to be *œdematous*.

The fibrine of inflammatory serous effusions remains in solution for weeks or months within the body, during life, but will coagulate readily when withdrawn. This delay of the fluid to coagulate within the body, is a propitious event. So long as it is liquid, absorption may still ensue without its undergoing any ulterior change when the inflammation subsides. This subsidence of the inflammation, however, is necessary, for it is known that so long as inflammation continues, there is impairment of function, and absorption does not take place.

One constant characteristic of the *productive* effects of inflammation is, that growing material is always developed from the pre-existing germinal elements of the tissue involved; and the effusion that results from mere mechanical obstruction to the flow of blood, is very different from the fibrinous effusion of an inflammation. In the former case, the fluid effused from the blood is merely the serous part, as the fluids of *anasarca* and *ascites*, and will not coagulate. Such fluids, as a rule, neither present fibrine, nor are any granules, particles, or cell-forms developed in them, from the elements of surrounding tissue.

Fibrine can only be made to exude upon any surface or part in a state of irritation or inflammation. Such a local change is suffi-

cient to cause the exudation of fibrine, independently of obstruction to the circulation; and the cause of the greatest differences in the nature of exudations, is to be found in the special constitution of the irritated parts (VIRCHOW).

It has been clearly shown (SIMON, LEHMANN, BEALE), that there are two essential characteristics of inflammatory effusion,—(1.) It tends to contain certain ingredients in larger proportion than that in which they exist in the blood—excess of chloride of sodium and of phosphates and albumen; (2.) Organic forms find in it a suitable place for growth.

The site of effusion resulting from inflammation, is important, as sometimes constituting the chief element of danger—a danger sometimes immediate, from the mechanism of the parts affected. A large quantity of fluid is often poured out in a very short time. The cavity of the pleura may fill in a few hours, and the lung may be compressed by it to a half or third of its bulk: and if both pleural cavities become thus affected, constituting *double pleurisy*, the patient must die from suffocation, if not at once relieved, by allowing free vent to the fluid. This operation is called *tapping* the chest, or, technically, "*paracentesis thoracis*." Serous effusion into the areolar submucous tissue of the glottis may also produce almost immediate death from suffocation, unless the cavity of the larynx is immediately opened to admit the air to the lungs (WATSON).

2. *Blood Effusions or Extravasations*.—Such chiefly occur from rupture of the new vessels developed in the newly formed material, which has just become vascular (ROKITANSKY). In the ordinary course of internal inflammations, extravasations of blood are rare, and betoken an unfavorable state of the constitution generally, such as occurs in typhus fevers, in scurvy, in purpura, or in syphilis. The post-mortem evidence of such extravasations is the presence of a colored cicatrix at the spot of rupture and effusion, and the color is found to be due to the presence of hæmatoidine, generally in the form of a mass of aggregate crystals, composed of minute rhombic columns, and which may be considered as the regular typical ultimate form into which hæmatine is converted in any part of the body where considerable masses of extravasated blood continue to lie for any length of time, *e. g.*, apoplectic clots and coagula in the Graëfian vesicle of the ovum after menstruation (VIRCHOW).

Mr. Paget correctly observes that we must not confound with hemorrhages the cases in which the inflammatory products are merely blood-stained, *i. e.*, have acquired a more or less deep tinge of blood, through the oozing of some of its dissolved coloring matter. The natural color of inflammatory new formations is grayish or yellowish-white, and even when they have become vascular, their opacity in the recent state prevents their having any uniform tint of redness visible to the naked eye. When inflammatory products present the tinge of redness, it is either because of hemorrhage into them, or because they have imbibed the dissolved coloring matter of the blood; and when this imbibition happens during life, or soon after death, it is important, as implying a cachectic, ill-maintained

condition of the blood, in which condition the coloring matter of the corpuscles becomes unnaturally soluble.

3. *Inflammatory Lymph or Fibrine*.—This product is so named to distinguish it from the lymph in the lymphatic vessels, with which it is probably not identical (PAGET). It is a characteristic primary product of the inflammatory process, and was called originally "*lymph*," or "*coagulable lymph*;" and more recently it has been called "*exudation*," or "*fibrinous*" or "*inflammatory exudation*." "It is at first," says Mr. Paget, "probably always a pellucid liquid exudation which passes through the bloodvessels"—"sweats through them," as Simon hath it,—"and especially through the capillaries of the inflamed part. Its most characteristic general properties are, that it may become a *nidus-substance*, capable of taking some share, or of assisting in promoting the growth of new elements like the natural connective tissue of the body." But the nature of the products of this development in the "*inflammatory lymph*" varies much, according to the part and the state of the constitution; and accordingly, Bennett attempts to distinguish the products of *simple exudation* from those of *tuberculous* and *cancerous* exudations. The typical elementary forms which may grow amongst inflammatory lymph vary according to a much greater variety of circumstances. The circumstances which tend to modify the type of the inflammatory process, or impart to it a particular *tendency* in respect to the nature of the productive material, may be shortly stated as follows:

1. The nature of the tissue in which the inflammation takes place.
2. The period at which the product is examined after exudation has taken place and growth commenced.
3. The state of the blood and the nature of the *zymotic* or *constitutional* morbid state which may be associated with the inflammation.
4. The amount of vascularity which the affected part retains.
5. The amount of the local exudation, and the extent of healthy tissue implicated.
6. The suddenness of the phenomena of exudation and of growth.
7. The persistence of the inflammatory state in its vicinity.
8. The amount of fluidity, serum, serous effusion, blood, or mucus, associated with the *inflammatory lymph*.

These are the chief circumstances which determine and modify the elementary forms which may grow amongst the "*inflammatory lymph*," and which may advance to further development, or to degeneration. The circumstances, severally, or more or less collectively, influence the different stages of progression, by which the local lesion of the inflammatory process may ultimately terminate in *resolution*, in *permanent organic mischief*, in *death* of the parts involved, or in *death of the patient*.

There are several typical forms, especially found growing amongst the "*inflammatory lymph*," and in the growth of which the material of the lymph may take some share—namely, the granular, molecular, or fibrillated development of *fibrinous* products and *corpuscular* forms.

Rokitansky describes these typical forms by the terms "*fibrinous*" and "*croupous*," and Dr. C. J. B. Williams by the names of "*plastic*" and "*aplastic*." Examples of each variety may illustrate the application of the terms. To the *fibrinous* or *plastic* variety belong the serous effusions already referred to, and perhaps also the granular, molecular, or fibrillated growths. The *corpuscular*, *croupous*, or *aplastic* forms of lymph are represented by those growths which never become consolidated, as in the early formed contents of vesicles in *vaccinia* and *herpes*; in the *fluid of blisters* raised in cachectic patients; in some instances of *pneumonia*; and in some forms of inflammation of *serous membranes*. In by far the larger number of inflammatory products these typical forms are mixed in various proportions; and the larger the proportion of corpuscles in new growth, the greater is the probability of suppuration, or of some other degenerative process, and the more tardy is any process of development into tissue, such as that of adhesions, indurations, and the like. In other words, the preponderance of granules, molecules, and fibrillated material in the new growth, is generally characteristic of the "adhesive inflammations;" the preponderance of corpuscles, or their sole existence in a liquid medium, is a general feature of the "suppurative inflammation." The hardness of inflamed parts is due to the former of these typical forms of inflammation, and is exemplified in the case of a *phlegmon* or *boil* before it suppurates; as also in a lung in a state of *hepatization*, when its textures are enclosed by lymph, "just as the stones of a wall are by the cement;" also in the hardening of a chancre.

On the surfaces of inflamed membranes the new growth forms a layer of a membranous firmness or consistence, to which the name of *false*, *adventitious*, or *pseudo-membrane* has been given. By this new growth the naturally opposed surfaces of parts which are inflamed are apt to adhere. This is commonly seen to be the case between such serous surfaces as the *pleura*, the *pericardium*, the *peritoneum*, or the *edges of a wound*. The inflammation associated with this organization is sometimes called "*adhesive inflammation*;" and Dr. John Thomson ascertained that this growth and organization might be effected between the surfaces of wounds in less than four hours after they were inflicted.

On the surfaces of mucous membranes may generally be seen the "corpuscular" typical form of new growth as a result of inflammation. It has little tendency to cohere, but grows in films, gelatinous masses, shreds, patches, or delicate casts of the surface upon which it was formed. The new growths in chronic catarrh of the intestines are an example; so are the membranes sometimes passed from the cavity of the uterus, and called *dysmenorrhœal membranes*. In the "adhesive" form of inflammation the new growth of granules or of molecules may ultimately assume the form of *fibrous tissue* interstitial to the textural elements inflamed. Examples of this organization are seen in the laminated and nodular thickening of the capsules of the spleen, the thickening and induration of the periosteum, or the capsule of the hip joint in chronic arthritis; and by virtue of the peculiar tendency to contraction which fibrinous prod-

ucts possess, the contractions of parts are to be explained which have been the seat of such a form of inflammation.

There are instances also in which the new growth assumes the form of adipose tissue, elastic tissue, and epithelium (KIRKES, VIRCHOW, PAGET); and bone is a very frequent ulterior change which it assumes, especially when the new growth is interstitial to fibrous tissue; but these ulterior events only happen *after* the inflammatory process has ceased in the part. So long as the inflammatory phenomena continue, the tendency of the new growth is to assume the *corpuscular* form, such as pus, rather than the more adhesive forms of organization. Hence rapid organization accompanies, as a general rule, a minor degree of action; and by depressing the action of a part, we tend to prevent the threatened occurrence of suppuration.

The existence of the inflammatory state, associated with an interstitial exudation, influences the simplest *corpuscular* forms of organization. *Lymph-cells*, or *simple primordial* forms, occur, which are represented by the corpuscles of chyle, lymph, the white corpuscles of the blood, and by those of granulations on the surface of a wound. These simple cells become developed amongst the lymph while it is still fluid, transparent, and apparently homogeneous. The first discernible organic form in the lymph of *herpes*, for example, is that of a mass of soft, colorless, or grayish-white corpuscles, about $\frac{2}{3000}$ th or $\frac{1}{3500}$ th of an inch in diameter, round or oval, pellucid, but appearing, as if through irregularities of its surface, dimly nebulous or wrinkled. It does not look granular, nor is it formed by an aggregation of granules; nor, in its earliest state, can any cell-wall be clearly demonstrated, or any nucleus on adding water. In a few hours, however, a pellucid membrane appears to have grown over its surface, permeable by water, which raises up part of it like a clear vesicle, while the contained mass retreats or subsides to the lower part of the enclosure, and appears more nebulous or grumous than before. A nucleus ultimately forms, and can be distinguished in this mass (PAGET).

From these primordial cell-forms in the lymph either the adhesive or more corpuscular forms of organization may proceed; and all the various forms of corpuscles described by authors as plastic cells, fibre cells, caudate cells, or fibro-plastic cells, and some forms of filaments, are developed from the germinal matter of the surrounding tissue by continuous development. Also, from the development of the primordial granules, corpuscles, or cells, all those elementary forms proceed which are known as "*pus-corpuscles*," "*granule-cells*," "*compound granule-masses or cells*," "*inflammatory globules*," and much of the *molecular debris-like matter* that makes inflammatory effusions turbid.

The modes of growth as well as of degeneration are well described by Mr. Paget, from whose work on Surgical Pathology the following examples and illustrations are chiefly taken:

1. The lymph may simply wither or waste, as may be noticed in the vegetations on the valves of the heart or large arteries when they become yellow, stiff, horny, elastic, and nearly transparent; or

in the lymph deposited over a compressed lung, associated with empyema or hydrothorax.

2. The fibrine of lymph may undergo changes similar to what is known as fatty degeneration,—changes similar to those which occur in the *primordial lymph-cell* when it is transformed into pus. The two changes generally go on together. To the former change, namely, the fatty-like degeneration of the fibrine, Mr. Paget gives the name of "*liquefactive degeneration*:" the solid fibrine of inflammatory lymph that becomes again liquid when suppuration takes place, as may be observed in a hard mass of inflamed texture when it becomes soft.

This is a degeneration which brings the new growth into a state favorable for its absorption, or to the *resolution* of an inflammation. Examples of such an absorption may be seen in *rheumatic iritis*, and the observations of Dr. Kirkes on the rarity of adhesions of the pericardium in comparison with the frequency of pericarditis may also be explained in this way.

3. Melanic degeneration of lymph and new growths is not unfrequent, as in *peritonitis*.

Concurrent with these degenerations of the lymph-granules and molecules are the degenerations of the *corpuscular elements*.

1. They may wither, as in the dried-up pus of chronic abscesses.

2. The fatty degeneration of cells is said to be shown in their transition to the *granule-cell*, known also as the *inflammatory globule* of Gluge, or the *exudation-corpuscle* of Bennett. The history of the formation or growth of these corpuscles is still doubtful.

The description of them, as originally given by Gluge, in describing the alterations of blood in inflamed parts, is as follows:

He observes "that the blood-globules lose their tegument and their color. Their inner substance alone remains, which, however, does not remain solitary; but by means of a whitish connecting material the masses become agglomerated, and form dense, opaque, round groups, containing on an average from twenty to thirty of the smaller bodies, which examined singly, are quite light and transparent. By means of pressure or acetic acid the associated granules break down into the individual bodies, and we see that the opacity is merely owing to the association. The associated bodies have a diameter in the mass of from $\frac{1}{80}$ th to $\frac{1}{30}$ th of a millimetre; the single granules are from $\frac{1}{80}$ th to $\frac{1}{40}$ th of a millimetre. These associated bodies," says Gluge, "I have seen in the bloodvessels, so that we have not here to do with a fluid which, transuding through the coats of the bloodvessels, is changed into granules. They escape by bursting the capillaries."

That this cell or corpuscle is formed within as well as without the bloodvessels, is apparent from an examination of inflammatory lungs or brain-substance. The corpuscles may be seen to coat the bloodvessels exteriorly and interiorly to their walls; and the formation of the corpuscle of Gluge can also be traced through stages of development, as described by Vogel, Bennett, Kölliker, Hasse, and myself; as well as through stages of degeneration from the normal state of some corpuscular elements (textural or morbid), the occurrence of

which has been described by Reinhardt, Dr. Andrew Clark, Dr. W. T. Gairdner, and Mr. Paget.

The essential ingredient of which the compound granule-cell is composed appears to be oily or fatty matter; and these cells vary considerably in their appearance, according to the fineness with which this matter is divided. In some the oil-drops are large, in others they are small and quite granular. They are by no means confined to inflammatory parts. Kölliker, in examining morbid products in an animal, has seen oval blood-discs included in these corpuscles, showing that the cell-membrane may be in some instances a subsequent formation in their progressive development. This view of their nature would imply that a number of the original oil or fluid granules come into contact with each other, and cohere into a glomerulus, which subsequently becomes invested with a membrane, and constitutes a cell, the contents of which gradually undergo some morphological process by which they are resolved, and ultimately pass into the circulation (SIMON).

My own observations on this point, published in 1849, and chiefly made upon inflamed pulmonary tissue, led me to express the same result, as to the nature of this compound corpuscular development, in the following statement:

"1. The formation of clear, transparent, non-nucleated cells may be observed.

"2. The formation of cells with a nucleus and nucleolus are seen, differing from pus-corpuscles in their large size, and in having a single nucleus. These are formed in the fluid of coagulated exuded matter, and become gradually filled by minute granules, which, when few in number, readily admit of the nucleus being seen. Subsequently, however, they conceal it; and the originally smooth cell-membrane becomes rugged, the granular cell appearing as a spherical agglomeration of granules. Subsequently the cell-wall appears to vanish, the enclosed granules to separate from one another, and to fall into irregular heaps" (*Edin. Med. Journal*, No. 178, for 1849).

The following are the general facts connected with the appearance of these corpuscles:

"1. They are formed in greatest abundance during the first stage of the exudation (the second stage of pneumonia, according to Laennec).

"2. As long as the capillary circulation is going on, and before complete stagnation has taken place.

"3. When the redness and condensation is the greatest, the corpuscles begin to disappear, or are not seen.

"4. They disappear altogether as the red softening passes into gray, becoming liquid.

"5. They are imperfectly formed, or not at all, in the deposits that occur during the progress of typhus fever or typhoid fever."

Associating these observations with the descriptions of Mr. Paget relative to the liquefaction of fibrine;—with those also of Zwicky and Gulliver, who found these corpuscles in the softened apex or centre of arterial clots;—with those of Simon, who states that they

are often found in the fibrinous clots of veins;—with their occurrence in the mammary secretion, in the softened parts of encephaloid cancer, in the vicinity of apoplectic effusions, and that generally they are extremely apt to be present where blood, or the products of exudation or secretion, are undergoing absorption;—does it not appear probable, moreover, from the lucid description given by Mr. Paget (when he says, that during the formation of these corpuscles “they present a gradual increase of shining black-edged particles, like minute oil-drops, which accumulate in the cell-cavity, and increase in number, and sometimes in size also, till they fill it”) that these compound granular cells, when associated with inflammatory products, fulfil a very important function, as the media through which the liquefied, softened, and disintegrated products of inflammation are gradually absorbed?

The observations of Reinhardt, Dr. Andrew Clark, Paget, and Gairdner also place it beyond a doubt, that compound granular cells may result from a fatty degeneration of the textural cells of a part; just as calcareous or pigmental degenerations occur, and which are common to primordial cells. While there can be no doubt, therefore, that fatty degeneration of lymph or textural elements may lead to the appearance of compound granular cells, that process can scarcely be called degeneration which is associated with development, growth, and complete absorption, by which the indurated and confused parts of an inflammation, such as the solidified portions of a lung in pneumonia, are ultimately cleared up.

Degenerate products are usually persistent, but the compound granule-cell is not. It seems to have an important function to perform in the removal of fluid, effete, or softened exudations, after which it too disappears.

The most frequent and important result of inflammation is the formation of pus by the growth of pus-cells. If a phlegmon or boil be observed, when it is a firm, hard, and solid mass of texture and exudation, we may feel in a few days that the solid mass has become fluid, and that it has not increased in bulk. The solidity and hardness are due to the inflammatory changes and effusion, the softening is due to the growth of pus-cells developed from the germinal elements of surrounding tissue (VIRCHOW, BEALE). So it is with the cells of vesicular eruptions which become pustular. The new cells there also become pus-cells—a change which may be accomplished in twelve hours, or sooner (PAGET). The following circumstances point to the development of pus from pre-existing germinal matter, namely, that,—(1.) A preliminary lymph-cell cannot always be discerned; (2.) The modification of the suppurative process, which occurs in the inflammation of mucous surfaces, where the formation of pus seems at once to take the place of the natural cell-growth, without any apparent distinction or alteration of the membranes of the mucous cells, corresponding in this instance to the most simple idea one can have of what Virchow terms *parenchymatous inflammation*, as described at page 93. Ultimately the natural mucous secretion undergoes a change. The characteristic cells on its surface drop off in all stages of abortion. Impaired co-

hesion of parts results,—an invariable expression of the inflammatory tendency. The epithelial covering becomes less characteristic, and gradually declines to small and simple cells, mingled with many primordial cells, which appear to have been hurried from the surface before they had time to undergo their legitimate development into the perfect mucous cells. From this sketch of what occurs, “it will be obvious,” as Mr. Simon writes, “that the anatomical distinction between pus and mucus must be as useless as the so-called chemical tests. Infinite gradations between the two destroy all practical value in such criteria. Mucus, as a copious fluid secretion, has no existence in health: the only natural secretion of a mucous membrane is its epithelium, which ought not to exist in quantity sufficient for any evident discharge. If the secretion be hurried, it immediately begins to assume the forms and physical characters of pus, even to the splitting of its nuclei with acetic acid.” In short, the essential process of inflammation has been established in the very cell itself, by the abnormal nutritive morphological relations which take place between it and the blood in the processes of life.

Inflammations of mucous membrane with a *mucinous* exudation (quite as characteristic of inflammation as *fibrinous* exudations) appertain to certain organs, *e. g.*, the gastric catarrhal inflammations. Such mucus is loaded with *mucin*, as a characteristic product of the inflamed mucous membrane, and which gives the tenacious, stringy character to the discharge.

Between healthy pus and healthy mucus there can thus be no confusion; but there are conditions between the two which yield neither “*praiseworthy*” pus nor healthy mucus.

Formation of Pus—Suppuration.—Well-formed, perfectly elaborated pus is a smooth, viscid, yellowish or cream-colored fluid, specifically heavier than water, averaging generally about 1.030, having little or no smell, and of an alkaline reaction. Microscopically, it is seen to be composed of certain essential constituents, namely, the pus-cell, and often minute clear particles, which seem to have some relation to the pus-cells as rudiments or nuclei of them. These constituents float in a fluid or serum called the *liquor puris*. The *pus-cells* are about $\frac{1}{25000}$ th to $\frac{1}{30000}$ th of an inch in diameter, pellucid, filled with semi-fluid albuminous contents, and sometimes containing a few minute oil-globules, which give the cells a granular appearance. Their shape appears to depend upon the density of the *liquor puris*. Sometimes a distinct, circular, dark-edged nucleus may be seen in the paler corpuscles, and sometimes two, or even three particles, like a divided nucleus. The minute clear particles often seen are not more than $\frac{1}{100000}$ th of an inch in size. Such are the components of *good, healthy, or praiseworthy* pus—the *pus laudabile* of the older authors—literally, the *pus to be commended*, as showing a benign form of inflammation, indicating that the process, though a morbid one—a disease—is going on regularly, and promises a fortunate issue (WATSON). It is the *laudable pus* of surgical writers. When, however, the process deviates from the state of health—deviates from the usual and regular course of the morbid action in a person otherwise healthy—then we find not only variations in the

pus-cells, but multiform mixtures of withered cells appear, with molecular and fatty matter, escaped and shrivelled nuclei, blood-corpuscles, and fragments of granular matter like shreds of fibrine. The *liquor puris* becomes unduly liquid, and the pus is then said to be *watery* or *ichorous*. It may even, in weak and tuberculous patients, consist chiefly of a thin serum, mixed with flakes or curdled, when it has been called *serous pus*. When the coloring matter of blood is mixed with it, it is called *sanious pus*. Chemical or vital changes of various kinds bring about a peculiar decomposition in pus while yet in contact with living parts, although it is probable that atmospheric air, or gases from an internal cavity, may have to do with the change; but hydrosulphate of ammonia is frequently developed, especially in abscesses about the alimentary canal, near the tonsils or the rectum. The stench is then most offensive when the fluid is set free. Pus, besides possessing certain chemical properties, may possess certain specific properties: thus it may be impregnated with certain poisons, as that of syphilis, or of small-pox; it is also often, in certain constitutional states, loaded with foreign matters, such as urate of soda.

The formation of pus is termed *suppuration*. It takes place under three conditions; namely,—(1.) Circumscribed; (2.) Diffused; and (3.) Superficial.

As examples of the circumscribed formation of pus may be mentioned an *abscess*, a *boil*, or *phlegmon*, in which the suppuration is inclosed within a cavity whose walls are composed of connective areolar tissue, and into which interstitial exudation of inflammatory lymph and serum has extended over a certain area. It happens that while the central portion of an area has become purulent (*i. e.*, has produced pus-cells as a result of the continuous premature proliferation of tissue), the peripheral part has maintained its firmness and solidity by activity of nuclear growth—and sometimes a “thin, opaque, yellowish-white layer, easily detached,” separates the suppuration area from the denser part. This has been called a “*pyogenic membrane*,” from the supposition that its function is to secrete the pus, whereas the nuclei and cells of the denser part are growing by continuous but premature development into pus-cells. Abscesses are sometimes formed without any of the usual accompanying signs of inflammation being present. They are generally slowly formed, and are named *old* or *chronic* abscesses. When suppuration happens in the natural cavities of the body it is still circumscribed. It is not then, however, called an *abscess*, but a *purulent effusion*.

Diffuse suppuration is exemplified in *phlegmonous erysipelas*, or the *purulent infiltration* of an organ. In such cases the inflammation extends through a wide extent of tissue, and from first to last the boundaries are ill-defined. The growth of pus-cells is distinctly interstitial. They are generally rapidly formed, and the tissue becomes thoroughly infiltrated, as if soaked in pus. The usual want of cohesion in the elements of tissue involved in inflammation prevails from the first, and ultimately large *sloughs*, or death of portions of texture, may take place. In some textures of a loose kind it is believed that the pus may spread about or infiltrate

parts by its own gravity, thereby leading to secondary destruction of tissue and the formation of what are called *sinuses*.

The incipient progress of diffuse suppuration is probably not dissimilar to that of a phlegmonous abscess, but the inflammation is generally of a different type, and all the processes are less complete; thus, no fibrinous lymph circumscribes the limits of the abscess, nor does any membrane form to limit the pus. The process of suppuration is less perfect, so that the abscess often contains shreds, or even large portions of mortified and loose connective tissue. The pus is less healthy, is thinner, containing a larger portion of serum, and oftentimes portions of loose fibrinous lymph. The *pointing* of this form of abscess differs also from that of the phlegmonous abscess, for the pus readily passes from its original seat, by infiltration, and, gravitating towards the most depending position, presents a soft, broad surface, without any indications of *pointing*. Such collections of matter are always of greater extent than phlegmonous abscesses, for the free transmission of pus from part to part occasions a great extension of the original disease. When these diffused abscesses open, the phenomena which result depend very much on the nature of the opening, and how it has been effected. "I have seen," says Mr. Hunter, "large lumbar abscesses open of themselves on the lower part of the loins, which have discharged a large quantity of matter, then close up, then open anew, and so go on for months, without giving rise to any disturbance; but when opened, so as to give a free discharge to the matter, inflammation has immediately succeeded, fever has come on, and, from the situation of the inflamed part, as well as from the extent of the lesion, death in a very few days has been the consequence." The same result has also occurred from liberating collections of the diffuse suppurative process in other parts. In erysipelas, however, which so often gives rise to this form of abscess, a free opening is often necessary, to allow of the escape of the portions of loose areolar tissue they contain.

Superficial suppuration may be observed in *gonorrhœa*, *purulent ophthalmia*, and generally in *inflammation of mucous and cutaneous surfaces*; and the growth of pus can be clearly traced where stratified, as well as columnar, epithelium naturally exists. Upon the skin the development of pus may be seen to proceed from the *rete Malpighii*, as a growth by continuous premature development of cells from this part of the young cuticle. In proportion as these young cells give birth to younger germs (proliferate), a separation of the harder layer of epidermis ensues, and a vesicle or pustule is the result. The exact spot where the growth of pus occurs, corresponds to what would be the superficial layer of the *rete Malpighii*; and if the membrane of the vesicle be stripped off, the cells of the *rete*, in process of conversion into pus, in place of epithelium, will adhere to the epidermis, and be stripped off with it (VIRCHOW). In the deeper layers, the cell-elements, which originally have only single nuclei (centres of nutrition, growing or germinal centres), divide, so that their nuclei (or centres of growth) become more abundant. Single cells have their places taken by several, which,

in their turn again provide themselves with dividing nuclei, and so the process of multiplication goes on.

Dr. D. R. Haldane, of Edinburgh, has observed and recorded the continuous development of pus-cells from the cylindrical variety of epithelium. In a case of small-pox, he found the larynx and trachea coated over with a soft, dirty-looking deposit, which was found to consist of pus-cells. On gently scraping the surface, the cells were found enlarged, and, in place of containing a single nucleus, each contained several—three, four, or more. These were derived from the proliferation of the original nucleus. External to the cells were young ones in all stages of development (*Edinburgh Medical Journal*, Nov., 1862, p. 439).

The more completely the epithelium is of the stratified kind, the less is the surface liable to ulceration (*e. g.*, the urethra in gonorrhœa); but those mucous surfaces where the epithelium is of the cylindrical form scarcely ever produce pus without ulceration (*e. g.*, the intestines). Pus-cells, mucous cells, and epithelial cells, are now regarded pathologically as equivalent elements, and which may replace one another; but physiologically they are not equivalent elements, inasmuch as they cannot perform each other's functions. Deeply seated pus-formation may proceed from *connective tissue*, or from the nuclei of vessels or sheaths of tissue. An enlargement of the *connective tissue* germs occurs (OTTO WEBER), which divide and subdivide, and so multiply excessively, by divisions of the larger germinal masses or cells. Round about the irritated or inflamed parts, where single cells lay, masses or groups of cells are formed, a large new formation grows, and towards the interior of this growth, heaps of little cells accumulate. These little accumulations occur at first as diffuse "infiltrations" of roundish masses, encircled by an intermediate growth, which continually liquefies as proliferation of the cells extend. Virchow regards this liquefaction as of a chemical nature; the intermediate substance (which yields gelatine) becomes transformed into mucus, and being ultimately converted into an albuminous fluid, is thus rendered liquid. Thus two different modes of pus-formation are distinguished, according as (1) the growth of the pus-cells proceeds from the germs of *superficial* tissue, like *epithelium*, or (2) from *connective tissue*; and two forms of inflammation can in like manner be separated from each other, namely—(1.) The *parenchymatous inflammation*, where the process runs its course in the interior of the tissue-elements (*e. g.*, connective tissue cells or germ masses, hepatic cells), without our being able to detect the presence of any free fluid which has escaped from the blood, but where softening and fluidity is due to the process above described. (2.) The *secretory* (exudative) *inflammation* of superficial tissue-elements, where an increased escape of fluid takes place from the blood, and conveys the new products of growth and altered secretion along with it to the surface.

The *parenchymatous inflammation* has from its outset a tendency to alter the elements of tissue and their special functions. Whereas the *secretory inflammation*, with a free exudation, in general affords a certain degree of relief to the part. Witness the relief which

follows the free flow of mucus in catarrh. It conveys away a great mass of noxious matter, and the part appears to suffer much less than a part which is the seat of a purely *parenchymatous inflammation*. In gonorrhœa also we have an example of how the pus resulting from the *secretory* form of inflammation is carried away by that transudation of fluid (exudation) which removes the pus-cells from the surface, without the slightest appearance of ulceration (VIRCHOW).

The description here given regarding the formation of pus is based on the great fact, demonstrated originally by Goodsir, that all new cells proceed from "centres of nutrition," from other cells, or from the nuclei of them; and, as Dr. Haldane justly observes,—“We must not expect to be able, in the case of every abscess or purulent discharge, to trace thus distinctly (as has been done in the preceding paragraphs) the origin of the pus-cells. There is only a certain stage in pathological as in physiological growth in which the actual mode of development can be followed. We might as well expect to be able to discover, by an examination of the mature foetus, the different steps by which its organs had been formed, as to be able, in a ripe abscess, to determine in what way normal had been converted into abnormal tissues.”

There are especially three events which, with more or less frequency, accompany or follow inflammation in a part. These are *softening*, *ulceration*, and *mortification*.

Softening, or diminished cohesion of tissue, is an almost constant result. It may be due not merely to mechanical separation by infiltration of the component elements of tissue, but to a loss of the vital cohesive properties, and impaired function of the tissues themselves, which tends towards their liquefaction and degeneration. Examples of this may be seen in the inflammation upon mucous surfaces already referred to, also in the inflammatory red softening of the brain and spinal cord, and in the lungs, where a peculiar brittleness and rottenness is imparted to their fibrous substance or skeleton texture. Such softening is due to vital changes in the proper tissue, often independent of any interstitial infiltration. The most remarkable example of inflammatory softening is that which occurs in bones. An acutely inflamed bone is so soft that it may be cut with a knife (STANLEY, PAGET).

But while some parts are *softened*, others are removed altogether by the process of *interstitial absorption*, as it has been termed. This phenomenon is best seen in bones which have been inflamed. Such absorption gradually precedes the extension of the inflammatory process, and leads, in the case of abscesses, to their spontaneous evacuation, commonly called the "*pointing of an abscess*." The inflammation continues, and the growth of pus moves along in a definite direction, towards the cutaneous or mucous surfaces of the body in its vicinity; but as the integuments are generally the more prone to inflammation, it is probable that they thus become soft, and yield sooner than the mucous surfaces do.

Ulceration goes on in the following way, as seen on an open surface, such as a wound or sore. Three processes progress simulta-

neously in order to effect ulceration: (1.) An exudation of *inflammatory lymph* and *serum* surrounds the mass of young cells which constantly continue to grow and to break up (proliferation). (2.) Cells are thus continually growing on the surface, to be carried off by a fresh exudation. (3.) Liquefaction of the gelatinous interstitial material supervenes, and so destruction of tissue takes place continuously. Thus an ulcer forms.

Granulation is one of the modes in which a wound, or sore, or a part previously actually inflamed, heals. It is then said to do so by "second intention," and is always a reparative process. Granulation may occur with or without suppuration. The first mode is extremely common. The latter is occasionally seen in the healing of syphilitic maculæ and ulcers of the cornea, and Mr. Hunter conceives he once met with it in the union of a broken thigh bone.

Granulation is associated with an exudation of inflammatory lymph, into which old vessels extend, and new ones are formed. A new surface thus results, which is "granular"—the granule being a small conical tumor or growth, composed of a mesh of terminal loops, formed by capillary vessels shooting into the effused lymph. The figure and color of the granulation are determined by the state of the circulation; when that is feeble and inclined to stagnate, the granulation is broad, flat, and spongy, and either pale or of a livid hue; when, on the contrary, it is vigorous, the granulation is conical or acuminated, and of a bright-red tint (TRAVERS). The vessels prolonged into the granulation are more or less tortuous, and so numerous as to require a high magnifying power to exhibit their distinctness after successful injection. These vessels become contracted to obliteration as the period of cicatrization approaches. Granulation may take place from a surface, or from the sides of an abscess. If from the cutaneous tissue, the sore heals by a process of skinning; the skin always springing from the edges of the wound. Again, if granulations spring from the walls of an abscess, their opposite surfaces may unite. Granulations sometimes form with great rapidity. Mr. Hunter has seen, after trephining a patient, the dura mater strongly united to the scalp in twenty-four hours. Granulations, however, have not in all cases an equal disposition to unite. Thus the granulations of fistulous abscesses are little prone to adhere, their surfaces being often as difficult to unite as those of a mucous membrane; indeed, it is often impossible to produce adhesion except by exciting a considerable inflammation. A part having healed by granulation, uniformly contracts. This contractile force is so great that although the sore made by the amputation of a thigh is seldom less than seven or eight inches in diameter, yet the cicatrix left on healing is hardly more than an inch or an inch and a half. From this cause we find, in parts that have been the seat of abscess, a marked depression at the point of cicatrization.

The reproductive energy of parts which heal by granulation, however, is not great. It is rare that the original tissue is perfectly reproduced. No fat, for instance, is re-generated in ulcerated adipose tissues; a muscle being divided, unites by a cicatrix of connective tissue, no muscular fibre being reproduced; and a divided

cartilage unites by tough fibrous tissue, but not by a cartilaginous bond of union. The skin, when destroyed, may be reproduced as a good imitation, yet generally it is imperfect. After small-pox, the *rete mucosum* is either slow in forming or never forms at all, so that the cicatrix or *pit* remains whiter than natural. Neither the smooth muscular fibres, nor any of the glandular structures of the skin are formed in its scars; but its fibro-areolar and elastic tissues, its papillæ, and epidermis, are all well formed in them. The reparation of the mucous membrane is equally imperfect, the villi being always wanting. The reparation of a flat bone, such as the cranium, is so slow that ten, twenty, and even fifty years pass away before a small trephine hole is filled up with bony matter. In like manner a healed cavity of the lungs is always marked by a cicatrix of areolar tissue altogether different from the original structure; neither, as far as we know, is the proper tissue of the liver, of the spleen, or of the kidney restored. A nerve simply divided is united by nervous matter in about twelve months or more; and the union is quicker and better in all tissues if air is excluded from the healing of the part.

It is a rule of all cicatrices, that the newly formed part is harder and of greater density than the original structure. Muscle, for instance, unites by coarse, dense, connective tissue; tendon most frequently by a harder and less pliant, but not tougher tissue, and sometimes by bone; and bone after a fracture is a more compact substance, and contains more phosphate of lime than before the accident; but, notwithstanding this addition, the new bond of union is not so strong, nor the living actions so energetic, as in the original structure. For when the constitution becomes enfeebled by severe disease, of a scorbutic kind especially, an old sore has been known to open, and the ends of a once-broken bone again to separate. It is equally a rule that a part having been once inflamed, the liability of the part to that form of inflammation is greatly increased; and also when new membranes or tissues have formed, that these tissues are infinitely more prone to disease than the original membrane.

Mortification is the death of a part, and may be complete or incomplete. In the soft parts the former is termed *sphacelus*, and the latter *gangrene*; while in hard parts, as the bones, there is a somewhat similar distinction, namely, into *caries* and *necrosis*.

Mortification of the soft parts may be white or black in appearance, humid or dry. The mortified part has a black aspect, when the blood is extravasated through the walls of the bloodvessels into the affected tissues, giving to the part a purple or dingy hue, while to the touch it is soft, inelastic, and doughy. The mortified part may appear white when, by the action of cold, the blood has been driven from the part, which subsequently freezes perfectly white.

Humid mortification occurs when the blood transudes in a fluid state, and after its exudation separates into its constituent parts, so that the serum, set free, dissolves in it the red globules, raises up the cuticle in bladders, and forms what are termed, "*phlyctenæ*."

Air, generated by a process of commencing putrefaction, is not unfrequently contained in the *phlyctenæ*, and gives, to the finger touching the part, a sensation of crepitation.

Dry mortification is a rare disease, and has sometimes been caused by the ergot of rye, or other diseased grain, used as food, giving rise to the disease known as *ergotism*. In the year 1716, dry mortification appears to have been, to a certain extent, epidemic at Orleans, fifty cases having been treated at the Hôtel Dieu of that city. Dodard described it as beginning generally in one or both feet, with pain, redness, and a sensation of heat or burning, like that produced by fire. At the end of some days, the part became cold, as black as charcoal, and as dry as if it had been passed through fire. Sometimes a line of separation was formed between the dead and the living parts, and the complete separation of the limb was effected by nature alone, and in one case, the thigh separated in this manner from the body, at the hip joint. In other cases amputation was necessary. Mr. Solly has given an interesting case of this description, which occurred in the practice of Mr. Bayley, of Odiham. The patient was a child, three years and seven months old, from whom, by this spontaneous process of nature, both arms were removed above the elbow, the left leg below the middle of the thigh, and the right foot above the ankle joint, being a remarkable instance, in modern times, of this destructive disease (see "*Ergotism*," and *Med.-Chir. Trans.*, vol. xxii, p. 23).

The bones, the brain, the lungs, the liver, the spleen, and the kidney are all liable to *sphacelus* and *gangrene*; so are the different tissues, as the areolar, cutaneous, nervous, and serous. The muscles, tendons, aponeuroses, and bloodvessels are likewise all liable, but in a less degree.

Local and General Symptoms of Inflammation.

Redness, or at least increased afflux of blood, swelling, or at least increased textural productivity, pain, throbbing, increased sensibility, disorder of function, arrest and change of secretion, are the phenomena which are associated with the local morbid state, or with the textures in its immediate vicinity. Increased local heat under all circumstances is constant. This has been recently proved to demonstration by the ingenious experiments of Mr. Simon, and his colleague, Dr. Edmund Montgomery (*A System of Surgery*, edited by T. Holmes, M.A., vol. i, p. 42). If the local process of inflammation, however, is carried on upon a minute scale, or in certain tissues, one or other or more of these symptoms may be absent; if, on the other hand, the local process proceeds on an extensive scale, and involves important and delicate textures of vital importance, then we have much more unequivocal expression given, not only to local symptoms, but to complex morbid processes affecting the constitution generally. Of these the chief are:

I. Inflammatory Fever.—Of the *constitutional symptoms*, as they are termed, the most prominent are those which indicate "*inflammatory fever, symptomatic fever, sympathetic fever.*" These constitu-

tional symptoms are of the greatest importance, not only by indicating the nature of the disease, as when the inflammation is connected with an internal organ removed from sight and touch; but they are highly important as a guide to treatment. The premonitory symptoms of coldness and shivering, are usually very decided, but not of long duration. They are succeeded by a stage of reaction. The pulse is then hard and swift. There is thirst, and greatly increased heat of surface. The secretions and the appetite may not at first vary much from the normal state, but on the whole are diminished. Exhaustion and emaciation do not proceed rapidly. This fever is pre-eminently one of strong reaction and vascular excitement, and these characteristics may be said to constitute its *type*.

A most minute description of the disorder of the general frame by *inflammatory fever*, according to its effect on the *systems* of the body, is thus condensed from the account given by the late Professor James Miller (*Principles of Surgery*, p. 39):

(1.) *The Nervous System.* There are aching, dull pains in the loins and limbs, restlessness, and much discomfort. The will and the power of exertion are diminished. Anxiety or foreboding of evil is felt, and expressed upon the countenance. The head is generally hot, the face flushed, the eyes suffused, and the skin hot and dry. Special sensation is at first exalted, but afterwards the intellectual functions become more and more disturbed. Ultimately delirium is established, and coma may ensue. (2.) *The Vascular System.* The pulse ranges from 80 to 130, or more, and the heart's action is proportionally rapid. The pulse is hard, rolling like a cord below the finger, and yielding but little to its pressure; or an irregularity of movement in the artery may exist, and thus a thrill or jar is imparted to the finger. There is increased fulness, as if the vessel were itself enlarged, and held a larger quantity of blood at each impulse; the heart is acting not only more rapidly, but more powerfully than in health; and the circulation is truly accelerated. Frequency, hardness, thrilling, are seldom, if ever, absent; but fulness may be wanting, and the pulse may be small instead of full. This modification is chiefly observed during serious inflammatory action, affecting important internal organs situated in the abdominal region. Hence it is sometimes termed the *abdominal pulse*; the artery resembling a hard, thrilling thread, rather than a cord. This pulse always exists in connection with great nervous depression and debilitated though rapid cardiac action; to which circumstance its smallness is probably due. In affections of the brain, on the other hand, producing coma, the pulse is commonly slow and full; the suspension of cerebral influence appearing to diminish the rapidity, without affecting the force, of the heart's action. There are idiosyncrasies also to be taken into account. The pulse may be naturally slow or rapid—fifty or ninety; and this must be allowed for, when previous inquiry has satisfied us that the patient is the subject of such peculiarity. (3.) *The Respiratory.* Respiration is quickened; the breath is felt to be hotter than usual; and an oppression is complained of in the chest. (4.) *The Digestive.* The tongue may be loaded, white, and moist;

or the edges and central tip may be red and dry: the latter is probably the more frequent combination. (5.) *The Secerning*. The secretions and excretions in general are materially diminished. The bowels are constipated—mainly from want of mucous secretion from their lining membrane; the skin is hot and dry; the mouth is parched; the urine is scanty, high-colored, generally acid, sparingly aqueous, and holding much saline matter, with comparatively little urea, in solution. (6.) *The Nutritive*. Digestion is interrupted; so is assimilation; as the fever advances, so does emaciation; and strength is more and more prostrate.

The chilliness, often amounting to shivering, marks the date of the febrile disturbance; and rigors more frequently attend the commencement of spontaneous inflammation than of inflammation caused by external injury.

Regarding the constitutional state characteristic of inflammatory fever, some important general conclusions, especially insisted on by Dr. Alison and Dr. Watson, may be thus shortly stated:

(1.) It is to be observed that there is no fixed relation between the degree or intensity of internal inflammations and the constitutional fever attending them; nor is the fever always proportioned in its degree of violence to either the size or importance of the part inflamed. In some cases, writes Dr. Alison, where we are sure that we have had inflammation going on under our inspection, to extensive effusion of pus, the pulse has been feeble, the skin cool and damp, and the patient exhausted and faint on the slightest exertion; while in others there is high and more inflammatory fever, and in some of these the organ inflamed has been so to no extent, and its function comparatively little affected, but yet the patient has become comatose nearly as in typhus, and died so. Laennec makes an observation of a similar kind (*Edin. Med. Journal*, May, 1857); and Dr. Watson observes that the fever may be high and very strongly marked in that common complaint, the *quinsy*, *cynanche tonsillaris*, or *tonsillia*, which can scarcely ever be said to imply much danger. (2.) The situation, the extent, and the degree of the local inflammation being the same, the fever commonly runs higher in young and in plethoric persons, and in those of sanguine temperament, than under opposite conditions. (3.) Inflammatory fever is modified in its expression, and especially in the characters of the pulse, by the nature of the part which is inflamed. This has been already alluded to in regard to inflammations of the abdomen, where the action of the heart is depressed, and the pulse is changed accordingly, tending to death by asthenia; and also in regard to the brain, when the mode of death tends to be by coma, the pulse being slow, labored, and full. (4.) The type of the inflammatory fever is very much modified by constitutional circumstances, such as the previous habits of the patient, and whether any zymotic disease is associated with the local inflammation. (5.) The inflammatory fever undergoes a further change of type (*a*) when suppuration takes place; (*b*) when it continues long; and (*c*) when mortification or gangrene occurs to a large extent. (6.) The febrile state follows generally the local disease; but (7) there is also good reason to believe that the *pyrexial*

condition, and the condition of *inflammation* in a part, may be excited in some instances conjointly; or, at all events, their periods of commencement may correspond so closely that it is difficult to conceive that one is the effect of the other. Observations are much wanted as to the exact ranges of temperature, as measured by the thermometer, in cases of inflammatory fever, and so to verify or set aside such general statements (see Billroth, in *Year-Book of Syden. Society* for 1861).

When inflammation proceeds to suppuration, a severe paroxysm of shivering is often the first indication of the formation of the pus, and the character of the fever undergoes a great alteration from that just described. The degree of the fever varies greatly even in this case, for a most copious formation of pus may take place from a mucous membrane, as that of the bronchi or urethra, and yet the constitution may hardly suffer in any appreciable degree; while a trifling amount of pus from a serous membrane will often be associated with fever of a fatal character.

In any case the character of a fever depends in a great measure on the constitution of the patient. If that be good, the fever is attended with a white tongue, with little tendency to become brown, also with much heat, and a full, strong pulse. On the contrary, if the patient's constitution be broken or impaired, the fever is of a low type—*asthenic*, as it is called. The event of *suppuration* is generally marked by a *rigor* of greater or less severity, while the fever hitherto has been *sthenic*. It is the occurrence of the *rigor* in the course of the inflammatory febrile state which gives it prominence and importance. It generally attracts the attention of the patient, and indicates to the physician that *pus* has been produced in the part or organ inflamed. As soon as suppuration is complete, and the abscess ripens, or pus approaches a surface to be discharged, and especially if any important organ is its seat, the fever tends to become *asthenic*, with a brown tongue and a rapid pulse, while the local pain in a great measure subsides. At this period the abscess must open spontaneously, or be opened by art, otherwise the patient may be in danger. The opening of the abscess, though attended with much pain from the contracting of the inflamed walls, is usually followed by relief of all the constitutional symptoms; the pulse rises, the tongue cleans, the appetite returns, and a visible and immediate amendment takes place. If, however, the patient has been exhausted by his sufferings in the earlier stages of the disease, the relief afforded is but transient, the pus degenerates into *sanies*, or is altogether suppressed, fever changes its type, and the patient sinks, too enfeebled to establish the reparatory process.

II. **Typhoid Fever.**—The type of fever known by this name is *asthenic* or *adynamic*. Feeble and more feeble the patient becomes; the pulse sinks; there is great impairment of the heart's action, and tendency to collapse; the features become pinched, shrunk, damp, and ghastly; and the skin is covered with a cold and clammy perspiration. Sometimes these *adynamic* characters may pass into that typhoid state in which nervous symptoms, such as delirium, somnolence, and tremors, prevail. These characters are known as *nervous*

or *ataxic*. The tongue becomes dry, black, and tremulous, sordes cover the teeth and harden on the lips and angles of the mouth. Low muttering delirium, stupor, or coma prevail; tremors affect the voluntary muscles, and the fæces and urine pass unnoticed. This form of fever sets in as a consequence of some untoward or unhealthy tendency of the inflammatory process, such as when mortification of the part occurs. Any cause, however, by which the system becomes extensively vitiated will bring about this form of fever. It is not necessary that the part should die. Putrescence of the infiltrated exudations in the inflamed part, degenerating and decomposing, poison the fluids circulating amongst them, and so, by absorption, may induce the typhoid state. If this happens with an internal organ, the event is generally indicated by a sudden cessation of all pain, at which the patient often appears very happy, and even joyous, while to the experienced physician its sudden cessation is assuredly an evil omen (Watson). The most important vital functions are deeply impaired by a prolonged existence of this type of fever. It tends to death by a complete sinking of the circulation, and diminution and loss of animal heat; or deepening stupor, with oppressed respiration, supervenes; or the patient dies by a combination of both conditions,—*asthenia* and *coma*.

III. *Hectic Fever*.—If suppuration continues beyond the powers of the constitution to supply the process with material to form inflammatory lymph and pus—if the inflammation continues, and becomes chronic as to time, inflammatory lymph continuing to be exuded, and pus continuing to form in profuse quantity, especially if an internal organ is its site—another type of *febrile* symptoms is apt to supervene, constituting *hectic fever*. It is not to be supposed, however, as was once believed and taught, that *hectic fever* is due, in every case in which it occurs, to the continued formation of pus. There are forms of *hectic fever* unconnected with suppuration anywhere, but associated with some analogous wasting of the bodily substance; for example, a prolonged secretion of milk in mothers who suckle their infants beyond the natural period. In all cases where a drain upon the system is established beyond its means, such a complex morbid condition of the body as *hectic fever* may be thus induced, and the mischief may not be revealed by any other symptoms. This type is particularly distinguished from the inflammatory and typhoid forms of fever by its remarkable intermissions, which are usually periodical; a period of remission and a period of exacerbation usually occurring once, and sometimes twice, in the twenty-four hours. It is also characterized by an excessive waste of the tissues of the body; and the sweating which attends the paroxysms causes great exhaustion. The assimilative and nervous functions are comparatively unimpaired, so that it is a febrile state generally of very long continuance. The mind remains perfectly clear—often vigorous and active—even when the body is debilitated; and if the intervals between the paroxysms are tolerably free from febrile excitement, the *hectic* type of fever may be protracted much beyond what at first sight might appear credible; and thus it is sometimes within our power to alleviate

greatly this condition. If, however, the fever does not abate during the remissions of the excessive paroxysm, when sweating continues profuse, and when suppuration or other wasting discharge is excessive, the fatal termination approaches rapidly.

The leading symptoms of this form of fever have been watched and described minutely by many observers, non-professional as well as professional. The fever creeps on insidiously, and almost imperceptibly; and the physician is at first led to suspect its existence by a very slightly increased frequency of pulse, and a small degree of heat of skin, occurring generally towards evening, and subsiding before the beginning of the next day. The pulse is subject to temporary quick excitement from slight causes, such as by exertion, by emotion, or by food, as after meals. The heat is especially felt in the palms of the hands and soles of the feet. The excitement of the pulse gradually begins to be more and more easily induced throughout the day; and towards evening the general exacerbation of the febrile state becomes regular, and is unmistakable. *Periodic* exacerbations or febrile paroxysms occur almost invariably towards evening, and remissions now become distinctly marked. The exacerbation reaches its height about midnight, and terminates by a profuse perspiration or sweating stage towards the morning. This sweating is sometimes called *colliquative*, and may be replaced or accompanied by *diarrhœa*. Occasionally a second paroxysm occurs in the morning after breakfast (Wood), or at noon, as described by Cullen; and as a mid-day meal was common in his day, it is probable that these slighter paroxysms may be attributed to such causes as the simple taking of food. Generally, however, in the earlier periods of this type of fever, the interval from morning till towards the afternoon and evening is free from fever; but in the advanced stage the fever becomes nearly constant, while the evening exacerbations and the morning sweats remain characteristic to the end. The pulse of the *hectic* patient is scarcely ever so hard and full as the pulse in *inflammatory fever*; nor is it so soft and compressible as the pulse of the *typhoid* patient. It expresses a middle condition between the two, of very variable character, both as to quickness and strength, according to the degree of exhaustion of the patient, and the amount of febrile reaction. Often during the paroxysm, or during temporary excitement from slight causes, it reaches 120 beats in the minute, the beats being performed with a jerk, as if the result of irritation upon a weakened heart (Wood).

The heat of skin during the paroxysm is often considerable, and always distressing, so that little more than the slightest covering can be endured. The respirations are quick and short. The appearance of the face is so characteristic that the *hectic flush* of the cheek, limited to a spot in its centre, is now well known. The delicate bright-red color and circumscribed form of the flushed spot contrast strongly and often beautifully with the pale cheek, and the bright and sparkling eye with its sclerotic of pearly whiteness. The surface of the skin is harsh and dry, and towards the close of life the region of the ankles is apt to become œdematous. The patient loses flesh rapidly, and as death approaches he becomes exceedingly

emaciated. It is then that *diarrhœa* is apt to supervene, and to aggravate the sweating, so as completely to exhaust the remaining strength. The mind, unclouded before, gently wanders now, and the functions of life cease, generally without a struggle. It is often one of the closing symptoms, most strongly marked, in pulmonary consumption; and the non-professional pen of our great novelist, Mr. Charles Dickens, has beautifully portrayed its more striking features in the death of Smike:

"But there were times, and often too, when the sunken eye was too bright, the hollow cheek too flushed, the breath too thick and heavy in its course, the frame too feeble and exhausted, to escape their regard and notice. There is a dread disease which so prepares its victims, as it were, for death; which so refines it of its grosser aspect, and throws around familiar looks unearthly indications of the coming change,—a dread disease, in which the struggle between soul and body is so gradual, quiet, and solemn, and the result so sure, that day by day and grain by grain the mortal part wastes and withers away, so that the spirit grows light and sanguine with its lightening load; and feeling immortality at hand, deems it but a new term of mortal life,—a disease in which death and life are so strangely blended that death takes the glow and hue of life, and life the gaunt and grisly form of death."

The forms of fever now noticed, as phenomena which may be associated with the inflammatory process, are to be regarded as various *types* which the febrile state may assume.

SECTION III.—DEGENERATION OF TISSUE.

Definition.—*Degeneration of tissue implies such a departure from the normal state as would give rise to a palpable appearance in its minute elements of a granular disintegration or detritus; or of any deterioration which, by the functional actions of repair in the normal state, could not have been left there, nor been visible.*

Pathology.—The circumstances under which degenerations occur are of the nature of decay and death. For example, degeneration occurs to an immense extent in the tissues of the aged, especially in the heart and arteries, and to a less extent in the voluntary muscles and the hard textures. Towards the close of the life of a part of the body, degeneration takes place—as, for example, in the textures of the placenta when utero-gestation is nearly complete. To such degenerations Virchow has given the name of *necrobiosis*, because death and degeneration seem to be brought about by altered life at the close of natural existence. In this respect it may be truly said, that "As we begin to live we begin to die." A spontaneous wearing out of living parts goes on, so that destruction and annihilation are immediately consequent upon life. *Softening* is the ultimate result of such degeneration, which becomes palpable chiefly by the decided friability of the parts. The minute elements of tissue lose their coherence, and at last really liquefy, so that pulpy or fluid products take their place. When it is remembered, also, how abundantly a gran-

ular fatty transformation occurs after death, the nature of degenerations becomes more intelligible; and my friend Dr. Lyons, Professor of Medicine in the Catholic University of Ireland, instituted a series of observations which beautifully demonstrated a process of morphy changes of tissues through dissolution and decay, till the mortal parts of our body return "ashes to ashes" and "dust to dust." To these morphy changes he has given the name of "*Histolysis*." To the same end are the demonstrations of Dr. Quain, regarding the conversion of muscle into fat, and of crude flesh generally into *adipocere*, accounting for the enormous fattiness of certain geological strata in which animal remains are abundant (MICHAELIS, quoted by SIMON). Such experiments and observations as those of Panum, Melsens, Ascherson, Gluge, Lyons, Simon, Burdach, Wagner, Michaelis, and others, and in which granules, vesicles, and cell-forms appear to arise spontaneously out of homogeneous albuminous fluid, will go far to explain many of the conflicting accounts which are given of the nature of the inflammatory products just described, and of the degenerations. Such forms may undoubtedly arise, as these observers show; and having arisen, they decompose and advance through changes such as Dr. Lyons has described under the name of *histolysis*. On the other hand, the *productive* results of inflammation undoubtedly grow from pre-existing tissue-elements, as already described.

All these degenerations are examples of *atrophy with* changes of texture (PAGET), as distinguished from *atrophy* resulting from simple decrease of bulk, the organ or tissue otherwise retaining its usual form, and to some extent its function. To recognize the following degenerations after death, the employment of the higher powers of the microscope is essential.

(a.) *Fatty Degeneration.*

Amongst the degenerations which are brought about by the spontaneous wearing out of living parts, the most widely spread, and the most important, is unquestionably the *fatty degeneration*. It is attended by a continually increasing accumulation of fat, which replaces the minute elements of tissue in different organs; and Simon concludes generally, regarding the presence of oil, or fat in textures uninflamed, that it is essentially a sign of weakness or of death, representing decomposition of effective material. In such *necrobiosis*, the elements of the tissue completely perish, and are replaced by fat-granules. Examples of this degeneration may be seen in the minute elements of muscle, especially of the heart; also in the acini of the liver, contiguous to the capillaries into which the branches of the portal vein break up; and in such degeneration the cells ultimately disappear, leading to loss of substance and atrophy of the gland; in the bloodvessels, in the *corpora lutea* of the ovaries, in the renal epithelium, and in many pathological products, such as pus, tubercle, cancer, and the like, when in process of decay; and, in short, in nearly all cell-structures, this degeneration is known to occur, except, perhaps, red blood-corpuscles and the elements of nerve-tissue.

In every texture the degeneration becomes evident in a similar manner. Isolated, extremely minute globules of fat appear in the cell-contents, and, becoming more abundant, they gradually fill up the cell-cavity. Usually the fat-granules appear at some distance from the nucleus; but ultimately they lie as close to each other as in the *colostrum corpuscles* of milk. At last the nucleus is no longer visible, and the membrane of the cell finally disappears—probably by a species of solution. If the degeneration occurs in the more rigid structures, as, for example, in the walls of arteries, the fatty granules retain the form of the cell which they replace. Such degeneration in arteries is first seen in the connective tissue corpuscles composing the innermost layer of the internal coat. Afterwards the intermediate substance softens, the degenerate fat-granule masses fall asunder, and the current of blood may carry away the particles of fat with it. Thus a number of uneven places (cicatricial-like loss of tissue) may be produced upon the surface of the larger vessels, without the appearance of ulceration (VIRCHOW).

In fatty degeneration of the substance of the heart, there is discoloration of its whole substance. It assumes generally a pale, yellow hue, with peculiar spots on the papillary muscles. Short, yellow streaks, which communicate with each other, are to be seen in the direction of the primitive fasciculi, and pervading the substance of the papillary muscles.

Yellow softening of the brain is a form of fatty degeneration, and this yellowness is due to the accumulation of finely granular fat. At every point where fatty degeneration attains a high pitch, great opacity will always present itself. A transparent part becomes opaque as in the cornea, where the fatty clouding marks the *arcus senilis*, described by the late Mr. Canton, in persons past middle life, and which has been regarded as an index to the existence of fatty degeneration of other more important organs, although the importance of the sign may have been exaggerated. In some form of Bright's disease, the uriniferous tubules become filled with fattily degenerated epithelium, which appears on the surface as opaque spots.

Additional examples of this fatty degeneration, are to be seen in the *fatty liver*, and in *mollities ossium*, atrophied renal capsules, and thymus gland, and the muscles—voluntary as well as involuntary—the fatty degenerations of the placenta, of cartilage, of bone, and of morbid growth: indeed, there is no kind of tissue, healthy or morbid, which may not undergo fatty degeneration.

When the normal structure of the part is thus transformed into fat, it is ultimately destroyed, and the place of the histological elements is gradually occupied by a purely emulsive mass—a kind of milk or fatty *débris*—that is, an amorphous accumulation of fatty particles in a more or less highly albuminous fluid (VIRCHOW).

With reference to fatty degeneration in particular organs, see the account given of local diseases.

(b.) *Mineral Degeneration—Petrification.*

The process which is followed by tissues undergoing this form of degeneration is very similar to that described in the previous paragraphs; but it is necessary to distinguish the forms of mineral degeneration as distinct from ossification. Formerly every kind of tissue condensed to the same degree as a bone was considered ossified, and the condition described as "*ossification*." But although a part may have lime in its intercellular substance, and although stellate cells may be present in it, yet it may be merely "*calcified*," or, as Virchow terms it, "*petrified*" tissue, and this condition he briefly describes as "*petrification*." Pathological ossification presupposes that the tissue or part which ossifies is called into existence by growth, and not that a previously existing tissue or part merely assumes the form or hardness of bone by absorbing calcareous salts. Ossification always begins by a growth of new tissue; and deposition of calcareous salts in its substance does not take place till a comparatively late period.

Calcification or *Petrification* is a degeneration comparatively more frequent in the peripheral arteries, and occurs most commonly in cases where there is a tendency to calcifications generally, and where calcareous salts are set free at other points in the system, to circulate with the juices (VIRCHOW). The lesion is to be distinguished also from *atheroma* of the arteries. In both conditions the artery may be felt to be a hard and rigid tube, with a calcareous feel to the knife or the touch. A careful examination microscopically will show that the degeneration is in the middle coat, that *calcification* or *petrification* of the minute muscular cell-elements has taken place, and that the fibre-cells of the circular fibre coat are transformed into calcareous spindle-shaped bodies. The degeneration may also invade surrounding parts, while the internal coat of the artery may be unchanged. The larger arteries are often brittle, from the mineral degeneration of their tissue—associated often with fatty degeneration (*atheroma*). Patches or plates of the mineral substance may be seen imbedded in the middle coat after the inner membrane is stripped off. When the smaller vessels undergo the mineral degeneration, the deposit resembles particles of oil; and the nature of such an appearance can only be determined by the application of mineral acids, which will dissolve the mineral matter with effervescence.

Nerve-cells, the fibrous membrane of the brain, the pia mater, and the choroid plexus, are all liable to undergo the mineral degeneration. Exudations and new growths are similarly liable. Dr. Bennett has seen the gall-bladder converted into a calcareous shell, and the pericardium into an unyielding box of mineral matter inclosing the heart. The cardiac valves are thus often covered with mineral incrustations. Cancer and tubercle growths may be transformed by the mineral degeneration; and Dr. Bennett has shown how the calcareous transformation of tubercles is the natural mode of arresting their advance.

The degeneration may follow upon the metastasis of calcareous salts, not excreted by the kidneys, in cases of caries of the bones, necrosis, or osseous cancer. I have seen specimens in the most interesting collection of Professor Virchow which show that metastatic deposits of bone-earth have taken place in the lungs and in the stomach under such circumstances. Considerable portions of the pulmonary tissue were *calcified* or *petrified*, without any apparent injury to the permeability of the respiratory passages. The lesion in the lung looked like a portion of fine bathing sponge. The mucous membrane of the stomach was in like manner transformed into a *calcified* or *petrified* mass. It felt like a rasp, and grated under the knife, so that the stomach-tubes seemed imbedded in a stiffened mass. The basis of such degeneration, in which the lime-salts find a resting-place, are the fine fibrous or connective tissues; and hence the degeneration is seen to occur in fibrous tumors, in serous membranes, in the parenchyma of lungs and stomach (as in the instance just mentioned), in cicatrix tissue on the skin, in the valves of the heart, in the connective tissue of muscle sheath, as well of the heart as of common muscle; in the tunica albuginea, in the fibrine coagula, in the heart's cavities, in aneurismal sacs, and in the thyroid and pineal glands. The cretifications of fibrine, of pus, of tubercle, of cancer, of vegetations, of coagula, all pertain to this form of degeneration; and the process may be traced through all stages of progressive degeneration from the pulp-like condition to cement-like, compact, calculous concretion, as in the phlebolite of veins; also in the turbid, chalky, speedily condensing juice of the cysts of the choroid plexus, and the cell-incrustations of the pineal gland concretions, as well as in the calcification of *sarcomata* and cancers. With regard to the degeneration as seen in tumors, Mr. Paget describes two methods by which it advances, namely, a *peripheral* and an *interstitial* calcification. The former is the rarer of the two. In this form of degeneration the fibrous tumor is seen to be coated with a thin, rough, nodulated layer of chalky or bone-like substance. In the interstitial form the degeneration is interspersed throughout the tumor, and so arranged that by maceration a heavy hard mass is obtained, variously knotted and branched, like a lump of hard coral (Paget, *Surgical Pathology*, vol. ii, p. 139.)

(c.) *Pigment-Degeneration—Pigmentation.*

In this degeneration *pigment* takes the place of the minute tissue-elements, as fat or lime did in the previously described conditions. It is seen in mucus-corpuscles, as in catarrhal pneumonia, in the pulmonary epithelium, in the acini of the liver, in the epidermic tissue, in the corpuscles of the blood in ague and *melanæmia*. As in the former degeneration, so in this one, a distinction must be carefully made between fat-granule cells and pigmentation, for in both cases apparently the same image is offered to view.

The fat-granule cells appear as brownish-yellow corpuscles, but their individual particles have no positive color; whereas the pigment-cells contain unquestionable gray, brown, or black molecules

of pigment, which are opaque (VIRCHOW). The diagnosis between the two is important, as in the brain, for example, where both sorts of granule-cells, namely, pigment-cells and fat-cells, may exist side by side. The former points to apoplexy having existed, the pigment originating probably in a solution of the coloring matter of the effused blood, the fat to cerebral softening. Therefore it is of importance for the pathological interpretation of the diseased condition to distinguish between pigment and fat in the granular form. Such pigment or coloring matter is insoluble in potash and acids—even in nitric acid.

In mucus-corpuscles or catarrhal cells the pigment exists in the form of grayish-black granules. They give rise to the smoky gray spots which are brought up in great quantity in the sputa in catarrhal states of the pulmonary passages; and to an extreme degree where accumulating masses of proliferating epithelium take place, as in catarrhal pneumonia and in the phthisis of colliers, so well described by Dr. William Thomson (*Med.-Chir. Trans.*, vols. xx. and xxi).

In the condition known as *melanæmia* (which, like *leukæmia*, has cells circulating in the blood, having made their way into it from definite organs), the cells contain black pigment; in the latter case (*leukæmia*) the cells are colorless. In *melanæmia* colored elements are met with in the blood which do not belong to it (STIEBEL, VIRCHOW, SCHONLEIN, HEINRICH, MECKEL, FRERICHs, and TIGRI). These pigment-cells in the blood were first seen to occur in the history of melanotic tumors, and were supposed to be due to the passage of particles from the tumors into the blood. This is not yet verified by observation. On the other hand, it is to enlarged spleens pervaded by black pigment that the change in the blood is to be ascribed in such cases, the color being due to the absorption of colored particles from the spleen. The class of cases which are the most fruitful source of black pigment in the blood are those of malarious diseases, *e. g.*, *intermittent fevers*, and especially in persons who have been long afflicted with a considerable enlargement of the spleen. In such cases Virchow found in the blood of the heart cells containing such pigment; and the cells that bore the color resembled in size and form the colorless blood-corpuscles; but there were also other cells of an oblong form and nucleated, within which a greater or less number of large black granules were to be seen. It is in the more severe forms of intermittent fever that such pigment-degeneration occurs. Such pigment is seen to accumulate in the minute capillaries of the brain, attaching to the points of division of the small vessels, and sometimes associated with the comatose and apoplectic forms of intermittent fever. Such pigment is also seen in the minute hepatic vessels (FRERICHs), where it ultimately gives rise to atrophy of the parenchyma of the liver. In a specimen of liver preserved at the museum of the Military Medical School, a deposition of melanotic pigment in a granular form is visible amongst the interlobular connective tissue, following mainly the course of bloodvessels in an irregular manner; and this case, like all the others yet recorded, was associated with a large black spleen.

The contamination of the blood in these cases seems due to a degeneration commencing in the spleen.

In post-mortem lesions, the textures are thus seen to be very variously tinted, red, yellow, brown, green, or black, generally resulting from chemical alteration in the coloring matter of the blood or bile. The red pigments, as a rule, are due to the altered hæmatine, originally of a yellow color; and which is the common origin of three different kinds of crystals,—(1.) Crystals of *Hæmatoidine* are the most frequent products of blood-degeneration (VIRCHOW). These are formed spontaneously in the body, out of hæmatine; and in their most perfect form present the shape of oblique rhombic columns, of a yellow-red color, or, in thicker pieces, of a deep ruby-red. In little plates it frequently bears a considerable resemblance to uric acid. In the majority of cases, the crystals are of extreme minuteness—difficult to resolve, even with a power of 300 diameters. They are insoluble in alcohol, ether, dilute mineral acids, and alkalis; and exhibit a peculiar play of green, blue, rose-tint, and yellow colors, under the action of concentrated mineral acids. If large masses of extravasated blood continue to lie for any length of time, this is the substance into which the blood is transformed. An apoplectic clot in the brain, for example, is repaired by a large portion of the blood undergoing this transformation, and the color of the resulting cicatrix is due to the crystals of *hæmatoidine*. When a young woman menstruates, also, the cavity of the Graëfian vesicle, from which the ovum escaped, becomes filled with coagulated blood, and ultimately *hæmatoidine* crystals are the last memorials of the event (VIRCHOW). *Hæmatoidine* is also allied to the coloring matter of the bile.

(2.) Crystals of *Hæmine*, arising out of hæmatine, differ from *hæmatoidine* in this, that hitherto they are only known as artificial products, which have not yet been seen in the human body. They are of a dark-brown color. (3.) Rectangular crystals or spicules of *Hæmato-crystalline*.

The yellow pigments are due to blood very much dissolved or dispersed, as in ecchymosis, or to bile, when it is absorbed in the blood, and tinges all the textures. Coloring matter due to bile may be recognized in the urine, by the play of colors it gives with nitric acid. A small quantity of acid gives a green hue; and, as more acid is added, blue, purple, violet, and a red or brown yellow color will ultimately appear. Of the brown and dark pigments, there are two kinds. One kind loses color on the addition of nitromuriatic acid or chlorine water; the other resists not only these agents, but even the action of the blow-pipe. This latter pigment consists of carbon. The former is a peculiar secretion formed within cells, or is a transformation of the coloring matter of the blood (BENNETT). Blue and purple pigments have been seen in urine containing *uroxanthin*, or the *Indican* of Schunk; and illustrate the close connection subsisting between animal and vegetable coloring matters (Parkes *On Urine*, p. 198). For much more interesting observations on the nature of pigmentation, consult Bennett's *Principles and Practice of Medicine*, p. 249.

Lardaceous or Albuminoid Degeneration.

This morbid process is one in which the normal textural elements of many organs and tissues are transformed into a peculiar substance, which has suggested, on the one hand, an alliance (in some respects only) with the chemical characters of lardaceous compounds, and, on the other hand, with albuminous substances similar to those which pervade the tissues of foetal life.

Professor Virchow, of Berlin, was the first to collect the facts regarding this peculiar form of disease, and to put them prominently forward. He proved the frequent occurrence in the animal economy of a degeneration, distinguished by the production of the peculiar substance to be described, which gradually takes the place of normal elements in the tissues so diseased. But Drs. Gairdner and Sanders, of Edinburgh, had anticipated many of the views and descriptions of the Berlin Professor, and, quite independently of Virchow, they initiated in this country the first steps in the elucidation of this very remarkable degeneration. They showed that the waxy condition of the liver and kidney was due to the same change as that which was seen to take place in the spleen. These valuable communications were made to the *Physiological Society* of Edinburgh; and an account of them may be read in the *Edinburgh Monthly Journal of Medical Science*, for February, 1854, p. 186, and also in May of the same year. Notwithstanding these researches, and those of Drs. Harris, Alridge, and others, in this country, we have much still to learn regarding (1.) The conditions under which this degeneration occurs; (2.) The forms in which it exists; and (3.) The symptoms of the lesion.

This degeneration has been long known by a variety of names. For many years the morbid anatomist has been familiar with a "bacon-like," or "lardaceous" infiltration of several solid organs of the body, and especially of the spleen and the liver. Portal and Abercrombie described the morbid condition in the liver as a "*lardaceous degeneration*;" and Hodgkin and Bright described the same disease as an "*albuminous infiltration*." In 1842 Rokitansky was the first to give a clear account, and to describe in detail the "*lardaceous*" infiltration of the kidney with an "*albuminous*" transparent substance. The lesion so described constitutes his eighth form of "Bright's disease." But Rokitansky made no chemical examination of the infiltrated material. He simply assumed, from its general appearance, that it was of an *albuminous* nature, and he rightly recognized its pathogenetic relation to certain cachexiæ. Budd has described the disease as "*scrofulous enlargement of the liver*." Oppolzer and Schrant have described the lesion by the name of "*colloid*," and Baron by the name of "*carnification*." The pathologists of this country have hitherto described organs so diseased under the term of "*waxy degeneration*."

[Recently Dr. W. H. Dickinson has proposed the term *depurative infiltration*, as being one of practical significance, and claiming kin with the suppurative process which he attempts to show is the most common cause of the condition.]

Such are the names, derived from appearances generally, under which the peculiar degeneration has been described, before microscopic examination demonstrated the structures implicated.

Chemistry and micro-chemical investigations have modified the views regarding the nature of the disease, and now and then have led to modifications in the nomenclature. Under this kind of inquisitive investigation it has been described, (1.) By Virchow under the name of "*animal amyloid*," he believing, from the behavior of the transformed substance with iodine and sulphuric acid, that the substance must be classified with the vegetable carbohydrates—cellulose and starch. (2.) Meckel retains the name of "*lardaceous*" or "*cholesterine disease*," believing that the essential character of the degeneration consists in the development of a peculiar fatty or lardaceous matter, of the nature of cholesteroline. (3.) The more extended and definite examinations by Friedreich and Kekulé have shown that the substance of the purest amyloid degeneration more closely resembles the *albuminous principles* than any other substance we know of; and (4.) Schmidt has arrived at the same conclusion; [and also Pavy (*Guy's Hospital Reports*, 1864).] The question, therefore, is not yet definitely settled as to the exact nature of the substance into which the tissues are transformed in the so-called *amyloid degeneration*, but the weight of evidence points to its being *albumen* in some form; and the albuminoid deposits in the spleen of children, so well described by Dr. Jenner, must be classed as examples of this degeneration, and probably also the special lesions in *rickets*.

Investigations relating to amyloid degenerations have taken especially two directions. Pathologists have endeavored, (1.) To trace the extension of the process of degeneration throughout various tissues and organs of the body; (2.) To determine the essential nature of the material into which the tissue is converted.

It was Professor Virchow who first turned the inquiry into the direction it has now taken, and which has given a remarkable interest to the micro-chemical investigation of the substance into which the minute elements of the tissues and organs are transformed in amyloid degeneration. Virchow stated that the large Malpighian sacculi in the spleen (which, in some instances, looked like boiled grains of sago), were sometimes composed of a substance which gave the chemical reactions of cellulose, as seen in plants. Cellulose and starch are both vegetable constituents—"isomeric" forms of some common material; and what gave special interest to the observation of Virchow was the discovery that cellulose is also an element in the covering or skin of the "*Tunicata*"—a genus of *acephalous mollusca*—and therefore not a constituent of only vegetable organization.

This discovery of cellulose in animal tissue induced Virchow to look for it or its analogue—namely, "starch"—in the human subject. He recognized it in the *corpora amylacea* of the brain. These contain a substance chemically related to starch or cellulose; and these bodies were first seen and named by Purkinje, who gave them the name they have, not on account of chemical characters,

but because he observed them to be laminated like starch. Of these *corpora amylacea* there are two kinds, namely,—(1.) Mineral bodies with concentric circles more or less soluble in mineral acids; (2.) Others which assume a blue tint with iodine, and a violet color on the subsequent addition of sulphuric acid. The relations of these two kinds to each other are still unknown. The first are the calcareous particles known as brain-sand; and *both* were at first described under the name of "*corpora amylacea*" by Virchow, which has led to some confusion. The term ought to be restricted to those bodies which, by physical and chemical characters, are assimilated to starch. The mineral bodies erroneously described as *corpora amylacea* are chiefly found in the cysts of the choroid plexus and in the pineal gland. On the other hand, the starch-like bodies have been found by Virchow, Rokitansky, Scherer, Kölliker, Busk, and other observers, in the *ependyma* of the ventricles, the *septum lucidum*, the *fornix*, the *auditory* and the *optic nerves*, and also in the *prostatic ducts*. Concentric lamination of these bodies is not always present; nor is the reaction with iodine and sulphuric acid constant. For these reasons Virchow began to examine those organs whose morbid state was described by the names already mentioned as having been given to the fatty or waxy spleen. He applied solutions of nitric acid, which, when hot, gave a yellow hue; he applied caustic ammonia, which gave a brown color; and from behavior with reagents generally, he concluded that the substance was "ALBUMINOID" in its nature. Iodine and sulphuric acid were subsequently tried. Iodine alone gave a strong yellow-red; sulphuric acid being added, developed a blue color, passing into a strong violet hue.* An excess of acid destroyed the violet hue, causing a dark brown-red color, passing into yellow. Meckel, subsequently to these observations of Virchow, came to the conclusion that there were four forms of this waxy material—that the basis of them all was a peculiar fat allied to cholesterine rather than to starch—that various saponaceous products are formed, ending in the development of cholesterine; and although he did not sustain his statement by anything like sufficient proof, he made the important discovery that it was the system of small arteries and capillaries which first suffered in this degeneration.

The inquiry into the chemical nature of the lesion becomes still more interesting when connected with the observations and discoveries of Bernard, Pavy, and others, on the "sugar-producing" functions of the liver, and on the material so formed, which may be separated by chemical processes, and has been recently shown by Dr. Robert McDonnell to be a substance which enters largely into the constitution of most of the tissues of the embryo (*Proceed. Royal Society*, vol. xii, p. 476). The results of these inquiries bring the "starchy substances" of animals in very close physiological alliance. The material so found has been called indifferently, "glycogene,"

* [The blue tint alleged to be obtained by the addition of sulphuric acid, is an error of observation arising from the fact that when sulphuric acid acts upon iodide of potassium, nearly always present in test solutions, iodine is thrown down which blackens the tissue.]

"amyloid matter," "zoo-amyline," or "animal starch." It owes its origin, not to any direct function of the organ, but its formation seems to take place almost immediately upon contact with albuminous matter, when this remarkable product is the result and which may be obtained as a white powder. It seems capable of being produced in greatest abundance by the hepatic tissue; but its formation may proceed at any part of the vascular capillary system. If, therefore, it is thus formed normally, it may also be formed, retained, or transformed in a morbid way. In *diabetes* we have an instance of the transformation of the product into sugar at the expense of the tissues at large; and which is so discharged by the urine.

The amyloid degeneration we are now considering has thus had various names to denote its presumed chemical nature, namely,—(1.) *Cellulose degeneration*; (2.) *Amyloid degeneration*; (3.) *Cholesterine disease*;* and now (4.) *Albuminoid degeneration*.

The analysis of the pure matter is very defective. Such as it is, it shows the substance to be *albuminoid*, and combined with *nitrogen* rather than *starch*; and those who describe the reaction of *cellulose* and *starch* with iodine and sulphuric acid, seem only to agree with each other in giving singularly diversified descriptions of color, which, perhaps, to those familiar with the writings of the late Dr. George Wilson, on color-blindness, may be accounted for. Such diversity may be explained in some measure, also, by the fact that the degree of concentration of the reagents materially concerns the results; for, as Virchow correctly observes, the blue coloration is only got after a considerable period, and in practised hands, and it may pass from a bright purple to a very blue color. Nevertheless, the action of iodine solution on the so-called amyloid tissue is peculiar and definite, independently of a blue color.

The appearance of a chemical reaction, which gives a hue different from the mere dyeing with the iodine, and which *suddenly* deepens in tone, from the moment it begins to take effect, to a deep brown-red color, is sufficiently characteristic. When this takes place with the solution of iodine ALONE, it distinguishes at once the substance from *cellulose* and *cholesterine*. The following tabular statement will show the differences more clearly:

CHOLESTERINE.	AMYLOID OR ALBUMINOID.
1. Unchanged in color by iodine alone.	1. Immediate coloration (of the nature of a reaction) by iodine alone.
2. Insoluble in water.	2. Dissolves in warm or boiling water. (Boil sections in a test tube.)
3. Melts with heat.	3. Does not melt with heat—only dries up, and still gives the same reactions with iodine.
4. Passes into a brown fluid on the addition of sulphuric acid concentrated.	4. Swells in, but does not dissolve with sulphuric acid, with change of color.
5. Soluble in ether.	5. Not soluble in ether.

* [Meckel first asserted that the deposit consisted essentially of cholesterine. This substance is not nitrogenous, and does not give the characteristic reaction with iodine, and has only been found in the liver in connection with the waxy deposit, and is, probably, an accidental associate, where fatty degeneration is coëxistent.]

By way of chemical analysis, very trustworthy results seem to have been arrived at by Friedreich and Kekulé. On submitting the white amyloid matter to ultimate analysis, they obtained the following composition in equivalents per cent. (*Med.-Chir. Review*, 1861, p. 59.)

	C.	H.	N.
<i>Amyloid</i> ,	= 53.58 . . .	7.0 . . .	15.04

Now, the composition of albumen, according to Dumas and Cahours, Lieberkühn, and Rüling, is as follows:

	C.	H.	N.
<i>Albumen</i> ,	= { 53.5 . . .	7.1 . . .	15.8
Dumas and Cahours,	{ 53.4 . . .	7.2 . . .	15.7
	{ 53.6 . . .	7.3 . . .	15.7
Lieberkühn, . . .	53.5 . . .	7.0 . . .	15.6
Rüling,	53.8 . . .	7.1 . . .	15.5

Surely these results show an almost perfect chemical identity between albumen and the morbid substance found in the so-called waxy spleen; and demonstrate that the waxy degeneration, in the spleen at least, is due to a peculiarly modified albuminous material, and not to starch? On the other hand, the chemistry of the corpuscular variety of the *corpora amylacea* occurring as a deposit in various parts—*e. g.*, in the brain, the prostate, and the *ependyma* of the ventricles—shows a reaction almost identical with starch. The corpuscles also have concentric laminæ, and, according to some, resemble starch-granules when polarized. As regards the corpuscles of the prostate, sugar has been chemically produced from them, and demonstrated by Trommer's test.

Many of these corpuscular varieties of amylaceous bodies are no doubt of the same nature as starch; and therefore the direction which inquiry ought now to take, will be to determine "Whether or not there is any chemical affinity on the part of the formless matter of waxy degenerations with the corpuscular variety of the amylaceous concretions?" Such an affinity has been assumed hitherto; but, so far as observation has gone, the evidence of any affinity seems to be getting less and less. On the other hand, the modifying effects of admixture and of growth, are very remarkable as regards these prostatic concretions. *Some* of them *iodine* will not color blue, not even after sulphuric acid has been added; and as growth proceeds, any amyloid matter they contain gradually disappears. Many admixtures of organic and inorganic substances give various shades of color; and the yellow-brown colored deposits failed to give forth sugar to Paulizky's attempts.

[Dr. W. H. Dickinson, after very elaborate investigations of the so-called amyloid substance, has reached the following conclusions: That the deposit essentially consists of an outpouring of a certain material which differs from the proper constituents of the body; that this material has no affinities with starch, but is essentially fibrous, and has been deposited in this form in consequence of the loss of the alkali with which it is ordinarily combined, and which seems to be necessary to hold it in solution—

4

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I.

On Chronic Bronchitis,

Especially as Connected with Gout, Emphysema, and Diseases of the Heart. Being Clinical Lectures delivered at the Middlesex Hospital, by E. HEADLAM GREENHOW, M.D., Fellow of the Royal College of Physicians, Consulting Physician to the Western General Dispensary, &c., &c.

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The purpose of this volume is to demonstrate the frequently constitutional character of Chronic Bronchitis, and its intimate association with many other diseases, in the relation either of cause or of consequence.

In the earlier lectures the author has endeavored to show, from the results of a large analysis of cases, and also from the history of many individual cases, in how small a proportion of bronchitic patients the liability to suffer from Chronic Bronchitis can be traced exclusively to catarrh, and in how large a proportion it can be referred to catarrh only in connection with one of three internal predisposing causes; namely, long-standing mechanical irritation of the bronchial membrane; some form of Dyscrasia; or previous illness of some other kind. The Gouty Dyscrasia is shown to be, of all others, perhaps the most fruitful source of Chronic Bronchitis. The fifth and sixth lectures deal with the subject of Pulmonary Emphysema; showing from an analysis of cases, and also from detailed cases, that Emphysema is frequently hereditary; that it is frequently found in connection with the Gouty Dyscrasia; and, lastly, that it is not unfrequently developed, in these circumstances, previous to the existence of Chronic Bronchitis. These facts, in the Author's opinion, prove the mainly constitutional character of Pulmonary Emphysema, and lead to the further conclusion that the degeneration of the tissue of the lungs which predisposes them to yield to mechanical causes of distension, in coughing or otherwise, is often a result of the Gouty Dyscrasia. In the seventh and eighth lectures, the relations between Bronchitis and Diseases of the Heart are fully considered. Bronchitis is shown to be a frequent sequel to diseases of the left side of the heart; whilst it is, on the other hand, itself a direct cause of disease of the right side of the heart.

The practical conclusion suggested by the work is that the first step towards the successful treatment of Chronic Bronchitis must be the discovery, and, in so far as may be possible, the removal, or alleviation, of the internal condition which, in so many cases, is the remote cause of the patient's ailment.

2.

The Pennsylvania Hospital Reports. (Published Annually.)

Volume 2, for 1869. Containing Papers by Drs. ADDINELL HEWSON, T. G. MORTON, DA COSTA, HUTCHINSON, E. HARTSHORNE, JOHN ASHHURST, JR., HUNT, PACKARD, AGNEW, HARLAN, TYSON, RICHARDSON, J. F. MEIGS, WM. PEPPER, &c., &c. Illustrated by lithographic plates and engravings on wood.

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"dealkalized fibrine." This is shown by the results of ultimate analysis of this morbid deposit, which make it agree in composition with fibrine and albumen. That it is fibrine and not albumen, is proved by its strong tendency to undergo contraction after its deposition. That it becomes converted into fibroid tissue, a metamorphosis which is common with fibrine whenever it is deposited in small bulk, as a coagulum in the arachnoid, or vegetations upon the valves of the heart. That in certain cases it is identical in appearance and reaction, as well as continuous in position, with the hyaline casts which are found in the renal tubes, the fibrinous nature of which it is not possible to doubt. A substance identical with it, and giving the characteristic color with iodine, can be made artificially out of fibrine by neutralizing or removing the alkali which the fibrine naturally contains. If potash or soda be added to the diseased tissue, thus artificially making a natural fibrine of it, it at once ceases to give the characteristic color with iodine. If a solution of sulphate of indigo be added to healthy tissue, the color of the solution is destroyed by virtue of the alkali contained in the tissues, but if the same solution be added to lardaceous degeneration, the color is vividly retained, because of the absence of alkali. An analysis of lardaceous liver shows a diminution by one-fourth of alkaline salts. While the alkalies are wanting in the morbid deposit, the earthy salts, as if to make up the deficiency, exist in larger quantity than in health (*Med. Chir. Trans.*, vol. i, 1867; *On the Pathology and Treatment of Albuminuria*, 1868).]

General Characters and Anatomical Description of Tissues which have undergone Amyloid Degeneration.—The cut surface of an organ so affected, has a semi-transparent appearance. It feels like a piece of soft wax, or of wax and lard combined (WILKS). It cuts into portions of the most regular shape, with sharp angles and smooth surfaces; and the thinnest possible slices may be removed by a sharp knife for microscopical examination, without any special preparation. The tissue is abnormally translucent. Water and alcohol, acids and alkalies do not produce any change upon the transformed parts, which may be kept for a length of time without decomposition. The organs affected are increased in volume, in solidity, and in weight, absolute and specific. Anæmia is predominant; but the color of blood or of tissue, shines through the semi-transparent morbid substance.

Amyloid degeneration is generally widely diffused; so much so, that a constitutional state of ill-health seems associated with its production; and in cases preceded by a local disease, such as caries of a bone, the degeneration may be found in the adjacent lymphatic glands only (BILLROTH). This is the earliest appearance of the degeneration yet recognized.

The small vessels of the tissue—the more minute arteries in particular—are, as a rule, the first structures attacked. The coats of the arteries become thickened and granular, and at last pellucid, transparent, and hyaline. Their calibre is reduced, and their cut section remains patulous.

It is the middle or muscular coat of the vessels which first changes. Each fibre-cell becomes a compact hyaline, pellucid, transparent particle, with an indistinct outline, and all the tissue involved becomes at last uniform, clear, and transparent. The degenerate

artery looks like a compact, homogeneous, silvery cord or thread, of a clear and glassy appearance, with a lustre like molten glass without polish, or like rough ice. This colorless, hyaline, degenerate tissue is very tough, but not hard nor brittle, like the calcareous degenerate parts. All other degenerations tend to obscure the original texture, by making it more opaque. This degeneration, on the contrary, renders the affected tissue more transparent and pellucid. It is in reality a *hyaline degeneration*. The specific cells of the functional parenchyma, when the degeneration affects a solid organ like the liver or kidney, next undergo the change, which finally spreads to the nutrient vessels amongst the connective tissue.

[The new matter as first observed, is transparent and homogeneous, and thickens the walls of the smallest arteries, with an apparent exaggeration of their transverse fibres; it soon penetrates the coats of the minute vessels and gradually works its way into the surrounding tissue. According to Dr. Dickinson the changes which result somewhat differ in the several organs. In the solid viscera the effused matter remains about the vessels, and fills the interstices of the structure. These organs, the liver particularly, often increase in size, and become hard, gray, and semi-transparent, as if intimately and uniformly infiltrated with white wax. The kidneys, supra-renal capsules, and lymphatic glands, are all apt to assume the same firmness and waxy translucency. In the spleen the deposit often exaggerates the Malpighian corpuscles, until they look like grains of boiled sago. The mucous membranes, which are very liable to the change, especially that of the small intestine, are differently affected. Their vessels are altered, but the exudation instead of being retained, as in the solid organs, seems to pass off, as a secretion, from their surface, giving rise to diarrhoea, or to vomiting when the stomach is the seat. Mucous membranes thus affected have an œdematous look and feel.

This morbid deposit may, though very rarely, appear as an independent growth, attached as a tumor to some part of the body. Dr. Murchison has described one instance (*Pathological Transactions*, vol. xiii), and Dr. Dickinson another (*On the Pathology and Treatment of Albuminuria*, p. 168). The tumors were of the size of a chestnut or plum, connected with the cerebral membranes, of a homogeneous, firm, translucent material, like cartilage, with a tendency to fibro-nucleated structure.]

When a solution of iodine is brought in contact with the affected part, a very deep violet-red color is produced; and this deep-red color seems to be *alone* a sufficiently characteristic test of the existence of the degeneration, especially when in a few seconds the color increases in depth from the moment it takes effect. It is a reaction which ensues between the iodine solution and the morbidly degenerate part. The best test-solution is composed as follows: *Twelve grains of Iodine* is to be dissolved with *twenty-four grains of Iodide of Potassium*, and mixed with *three ounces of Water*. Such a test-solution ought always to be at hand in the dead-house, or on making a post-mortem examination anywhere.*

* [The normal tissues take a yellow color with solutions of iodine; the morbid material a deep reddish brown, and it seems to have a strong affinity for the reagent, absorbing it readily, holding it tenaciously, and assuming its full color, while the healthy parts take only a faint and superficial tinge. If a few drops of the iodine

Elements of Tissue in which Lardaceous degeneration has been demonstrated.

1. Nervous System: ligamentum spinale cochleæ: atrophied parts of brain and spinal cord: gelatinous softening, and tumors.
2. Spleen: cells of the Malpighian sacculi and pulp: thickened walls of the arteries in all stages: the trabeculæ.
3. Liver: the hepatic cells and intralobular vessels (hepatic arteries): intercellular tissue.
4. Kidneys: Malpighian tufts and afferent vessels, the walls of which become enormously thickened: areolar tissue in the vicinity of the papillary ducts.
5. Muscular tissue of the heart and the uterus.
6. Bloodvessels of the villi and mucous membrane of the alimentary canal.
7. Osseous tissue.
8. Lymphatic glands.
9. Besides the original structures of the body, old deposits in serous membranes, having lost their fibrous character, becoming dense, more vascular and semi-transparent, undergo this metamorphosis (GAIRDNER).
10. Tubercle also becomes amyloid (GAIRDNER).
11. The cancerous nodules in a waxy liver also become amyloid (GAIRDNER).
12. In some cases of inflammation with exudation on the mucous membrane the exudation has assumed the amyloid degeneration (VIRCHOW).
13. The fibrine of a hæmatocele (FRIEDREICH).

The extensive range of organs in which this remarkable degeneration has now been demonstrated clearly shows that the lesion cannot be regarded as merely of local importance. Its occurrence seems rather to point to some general pathological state of which the degeneration is the expression. In the first instance it is found more particularly affecting the functional capillaries of the most important organs of the body—*e. g.*, the kidney, the liver, the spleen, the intestines, as well as the minute arteries of nutrition of those organs, and of the pia mater, bone, and lymphatic glands. The results of such a degeneration must therefore be sooner or later destructive,—(1.) To the function of the invaded organ; (2.) To its nutrition; and we can only arrive at a correct pathology of this degeneration by a close observation of the circumstances, condition, relations, and symptoms under which the lesion manifests itself. These must be studied especially in relation to the functional or physiological anatomy of the organs implicated. As yet the lesion has been recognized with certainty only in the dead-house. There it has been found associated with certain diseased states; and all the cases agree in this particular, namely, that the constitution of the patients, has

solution be poured upon a mucous surface or on the section of an organ, a uniform yellow tinge is procured; if this peculiar deposit is present, it becomes conspicuous by the contrast which its deep brown color presents to the unaffected parts (DICKINSON).]

been broken up by ill-health (cachexia) of some considerable duration before death. So it has been amongst the soldiers dissected at Fort Pitt and at Netley; and the following statement is a summary of diseased conditions, in the order which furnishes the greatest number of cases of amyloid:

Diseased States with which Amyloid Degeneration has been found associated, or upon which it is engrafted,—

1. *Diseases of the bones*, especially caries and *necrosis* in scrofulous subjects. *Rickets* also leads to the amyloid liver and spleen, as observed by Glisson, Portal, Rokitansky, Lambe, Læschner, Frerichs, and Jenner.

2. *Syphilis*, especially in its ulcerated forms, the cachexia having been prolonged. Syphilitic children have been the subject of it when newly born.

3. *The malarious cachexia*, especially *intermittent fever*.

4. *Mercurial cachexia* and *marasmus*.

5. Pulmonary and intestinal forms of *tubercle*.

6. *Albuminuria* and *anasarca*.

7. *Diseases of large arteries*.

As to the origin of the lesion or degeneration, Frerichs has propounded two questions, namely,—(1.) Is the lesion due to deposits from the blood of the amyloid, waxy, or albuminoid matter in some primordial form, and which is generated in the blood, in consequence of a local disease, such as caries of the bones? (2.) Is the amyloid or waxy matter developed locally in the affected tissue by the transformation or degeneration of the tissue into albuminous matter?

It has been more recently urged by Dr. Dickinson that the degeneration is always the result of extensive purulent formation of long duration.

[The views of Dr. Dickinson of the causes of this morbid deposit, as given in his interesting paper in the last volume of the *Medico-Chirurgical Transactions*, and his recently published volume on *Albuminuria*, are of practical value, and, in connection with his pathological researches, are worthy of careful examination. As far as the conditions which lead to the separation of this material from the blood, there is a loss both of alkali and albumen, the former at the expense of the fibrine, whilst the latter leaves the fibrine in relative excess. The researches of Dr. Dickinson would go to prove that by far the most frequent cause of the disorder is copious and long-continued suppuration, not necessarily connected with tubercle or any other constitutional taint, or with disease of bone. Of 66 tabulated cases which came under his notice, 51 were known to have been associated with a profuse and protracted drain of pus; while in 5, suppuration, though not under observation, had, apparently, from post-mortem appearances or other circumstances, gone on at some antecedent period. Thus five-sixths of his cases, indiscriminately taken, were associated with the purulent process. Believing in this undoubted relationship of cause and effect, the bond of connection is easily traced. The discharge removing the alkalies from the system, there is a relative increase in the amount of fibrine. Pus is an albuminous fluid rich in alkaline material, containing about one per cent. of alkaline and earthy salts, in the proportion of 10 of the former to one of the latter. With the fibrine relatively increased, and the alkalies positively deficient, the dealkalized fibrine is

deposited in the smallest arteries; the force of the circulation being lessened by distance, and broken by subdivision, the current has been sufficiently retarded to allow of coagulation or deposition. The deposit happening in the arteries and not in the veins, may be accounted for by the fact that venous blood is less rich in fibrine than arterial.

Albuminuria, especially when connected with nephritis, is the next most frequent circumstance of its occurrence. In 66 cases 4 were apparently due to this cause. A long continuance of albuminous urine must have an analogous effect to that of chronic suppuration. The albumen appears to carry alkali with it; albuminous urine, as a general rule, particularly in long-standing cases, is wanting in acid. In the 6 remaining cases there was no evidence of any morbid discharge, 3 of the subjects having been drunkards. Every process by which the blood is so altered as to contain excess of fibrine with deficiency of alkali is probably not yet known; other conditions unquestionably exist, which, impairing nutrition and altering the composition of the blood, may produce the effect of a chronic purulent discharge—the depurative deposit—without the medium of suppuration.

Dr. Dickinson's observations do not lead to the belief that syphilis has any direct power in the production of the deposit; nor is there any evidence that either cancer or tubercle are direct causes.]

Arguments are put forward by Virchow and Frerichs to show that the lesion may be due to a deposit from the blood; but the weight of evidence seems, on the whole, to point to a peculiar degeneration of existing tissue. (1.) In cases where the lesion follows affections of the bones, the lymphatic glands adjoining the diseased bones are implicated before the kidneys, liver, or mucous membrane of the intestines. (2.) General causes of ill-health (cachexia), pointing to impoverished blood, are in operation, and organs situated in different parts of the body are simultaneously affected. (3.) The fibrine of the blood itself has been observed to undergo the degeneration; for Friedreich found a substance which gave the amyloid reaction with iodine in the old fibrinous layer of the sac of a hæmatocele.

In this remarkable degeneration an acquaintance with a new fact in pathology must be recognized—*i. e.*, since 1854—associating itself with grave constitutional disease, and distinguished from every other morbid condition hitherto known, by the physical, chemical, and physiological characters just described.

The Clinical History of amyloid degeneration is remarkably deficient. The effect of the degeneration is to interfere with function of organs and nutrition of parts; and the injurious effects are the more marked as the lesion extends through many important organs. For example, hepatic cells cease to take part in the formation of sugar or the secretion of bile. Bloodvessels lose their power of transmitting fluid through their walls, and become impervious as to their canals. Hence those who suffer from amyloid disease have an appearance of *general ill-health*, denoted by paleness of the surface, by symptoms of anæmia, hydræmia, or by leukæmic affections of the blood; and the more so as the constitution is enfeebled by such morbid processes as ulceration of bones, syphilis, tuberculosis, or malaria. The sequence in which the different organs degenerate is uncertain. In most cases of caries and necrosis the kidneys seem

to be first attacked after the lymphatic glands. In cases of intermittent fever it is usually the spleen which is first affected; and generally it seems rare to find several, or all the organs affected to the same extent.

Signs or Symptoms associated with this degeneration discoverable during life.—On these points data are wanting upon which to found any statement. The pathological change is of so recent discovery that well-recorded cases, terminating in death, with verification of the symptoms by post-mortem inspections, are very few indeed. There is no subject, therefore, more full of interest, or one more likely to repay close observation and well-directed pathological inquiry, than the diagnosis of amyloid degeneration. Cases in hospital ought to be most carefully noted, and especially such ambiguous cases as those where marasmus, anæmia, or dropsy are *primary* symptoms, and which are not to be accounted for even after the blood has been examined microscopically during life, and the condition of the liver, heart, spleen, and lymph-glands carefully inquired into, without evincing signs of disease. In a remarkable case recorded by Friedreich and Kekulé, and quoted in the *Medico-Chirurgical Review* for October, 1860, *diarrhœa* and *vomiting* were of frequent occurrence, with a systolic murmur of the heart, and high-colored and albuminous urine, with a specific gravity of 1.019. The patient, a female, after suffering from tertian ague for twelve months, became dropsical and emaciated. The intestines throughout, the stomach, the colon, the jejunum, and especially the capillary vessels of its villi, were affected, as well as the vessels of the kidneys. The urine should be watched as to albumen, or deposits, and its amount in relation to body-weight should be recorded. When albumen appears, it goes on gradually increasing; and hyaline casts increase with the increase of albumen.

Dr. Stewart, of Edinburgh, records twenty cases, and *nine dissections* in cases of Bright's disease, where he considered lardaceous degeneration to have been present (*Edinburgh Medical Journal* for February, 1861). He records that large quantities of urine were passed in the early stage, of supposed waxy degeneration, and of a specific gravity from 1.005 to 1.015. In all the cases there was a striking general correspondence in the other symptoms; and Dr. Stewart thinks that from this similarity of symptoms, and from other considerations, he is warranted in believing that amyloid degeneration existed in the *eleven* cases that did not die. The history of all Dr. Stewart's cases is remarkably different from that of the fatty kidney which Dr. Bright figured in his first plate, and illustrated in his first case. Almost all of the cases were associated with long-continued wasting disease; and it has been long known that the form of renal affection accompanying phthisis, syphilis, and other wasting maladies, is the waxy or amyloid degeneration. Of the twenty cases related by Dr. Stewart, six were associated with phthisis, six with syphilis, two with caries, two with intemperance, one with cancer, one with chronic rheumatism, and two with no particular disease.

The degeneration is much more common than is generally sup-

posed. It has been observed very frequently amongst the soldiers who have been dissected at the Military Hospital for invalids, formerly at Fort Pitt and now at Netley. The microscope and iodine test can alone determine its absence; and without microscopic examination the absence of the degeneration cannot be determined. For a detailed account of "amyloid degeneration" in the various organs, see the description given under LOCAL DISEASES.

Before stating the principles which dictate the treatment of the complex morbid processes just described, and of individual diseases in particular, it behooves the student, first, to make a separate study of *the varying types of diseases, their prevailing peculiarities, and the constitutional tendency to change of type which they assume at varying intervals of time*; and, second, to observe and learn to recognize *the various modes by which diseases terminate fatally*.

CHAPTER IX.

TYPES OF DISEASE AND THEIR TENDENCY TO CHANGE.

IN describing, appreciating, or ascertaining the type of a disease, our attention must be directed to a variety of phenomena and conditions; and the type of the disease only becomes characteristic and distinctive when some one or other of those conditions becomes predominant, or manifests itself more decidedly than others. The hereditary or natural constitution of the individual may be regarded as an important element in determining the type of the disease. Town life, as compared with country life, also exercises an influence on the type of many diseases; and there are good grounds for believing that the town life and artificial habits of the present period are more prejudicial to the strength of the constitution than those which prevailed when large towns were but rural villages, and the inhabitants more simple in their mode of life, and less artificial in their habits.

The occupation of the individual in many instances exercises an influence over the complex processes of disease; and there cannot be a doubt that some diseases have altogether disappeared, while others have been so much modified that their resemblance to the original form or type can with difficulty be recognized.

With regard to Edinburgh and its vicinity, Professor W. T. Gairdner observes that the *changes of type* which have occurred in epidemic fever, and especially in typhus fever, during the ten years previous to 1862, or since the cessation of the great epidemic of 1847-8, are not less remarkable than the diminution in the amount of this class of cases. The *relapsing fever*, or *symocha*, which formed so large a part of the epidemics of 1843-4 and 1847-8, has for the time absolutely disappeared. *Typhus fever* has become less fatal to

those attacked than it was ten years ago ; while its general type, and some of its leading characters, have been remarkably modified. This is especially noticeable in the *diminished mortality* ; the *earliness of the appearance of the eruption* ; the *earliness of the crisis* (tenth to fourteenth day as a rule, and rarely prolonged into the third week)—a great cause of the diminished mortality ; for a day's delay of the crisis, in a case of any degree of severity, is an immense addition to the risk (*Clinical Medicine*, 1862, p. 156, *et seq.*). Nevertheless, it is the rule that diseases preserve their essential characters and nature from age to age, although the opinions of the profession respecting them and their treatment may change from year to year, or from one period of time to another. For example, *small-pox*, *measles*, *typhus fever*, *typhoid* or *intestinal fever*, *dysentery*, *diphtheria*, and the like, always remain the same as to their essential characters, and unchanged in their special symptoms ; but it must be remembered that they are also very often modified in their phenomena by the existence of such constitutional ill-health (*cachexia*) as may arise from *syphilis*, *mineral*, *vegetable*, or *alcoholic poisoning* of the system, as well as from *gout*, *rheumatism*, *scrofula*, *tuberculosis*, and *scorbutus*, from epidemic causes ; and especially the artificial mode of life in towns as compared with the more natural habits of rural villages. These are the diseases and the conditions of existence which are the main source of the deterioration of the human race, in all physical attributes, among such civilized communities as our own. Diseases also have arisen which appear to be more or less new to us, in some instances resulting from a hybrid combination of various pathological phenomena to be noticed presently. While this is undoubtedly the case, there is abundant evidence to prove that we now have in some respects a more healthful enjoyment of our life on the whole [although the constitution may not be so strong] than formerly ; and that the duration of man's life of late years has been, on the whole, prolonged. While it is the lot of "all men once to die," that final change is more frequently deferred than was wont, to beyond that period when, in the words of the inspired Psalmist, it is recorded that "the days of our years are threescore years and ten ; and if by reason of strength they be fourscore years, yet is their strength labor and sorrow."

It has been observed by a popular writer that there never were any specifics discovered against the *plague*, the *sweating sickness*, or the *leprosy*, and yet these diseases, so far as regards this country, are now amongst the things that were, and are almost unknown. They have disappeared, not before any marvels of medicine, or any perfection of chemical sciences, but as a consequence of the gradual amelioration of our condition through sanitary improvements. Observe, also, what sanitary science has done, in a comparatively short space of time, to ameliorate the condition of the British army. The Right Honorable the Secretary of State for War (Sir George C. Lewis, Bart.), in moving the army estimates for the year 1862 in the House of Commons, said,—

"Improvements have been introduced with a view to ameliorate the

social, moral, and sanitary condition of the private soldier. Much expenditure has been incurred for the sake of enlarging and improving barracks, and in carrying out various recommendations of the House of Commons with respect to barracks and the hospitals connected with them. I am happy to say," continues the Right Hon. gentleman, "that these efforts have not been unattended with important results, as will appear from authentic returns of the mortality in the service. These returns have been prepared by the Director-General of the Army Medical Department, and I believe they are perfectly authentic, though it is certainly difficult to believe that so great a change can have taken place in so limited a period. It is possible that the greater youth of some portions of the army may, to a certain extent, affect the returns, but I believe the difference is mainly to be explained by improvements in the sanitary conditions under which they are now called on to serve.

"DEATHS AMONG THE TROOPS SERVING IN THE UNITED KINGDOM ANNUALLY PER 1000 OF MEN.

	From 1830 to 1836.	1859 to 1860.
Generally throughout,	14	5
Cavalry of the Line,	15	6
Royal Artillery,	15	7
Foot Guards,	21	9
	From 1836 to 1846.	1859 to 1860.
Infantry of the Line,	17	8

"SIMILAR RETURNS FOR THE COLONIES ARE AS FOLLOWS:

	From 1837 to 1856.	1859 to 1861.
Gibraltar,	22	9
Malta,	18	14
Ionian Islands,	27	9
Bermuda,	35	11
Canada,	20	10
Jamaica,	128	17
Ceylon,	74	27

"I have other returns from other colonies," continues the Right Honorable gentleman, "and I believe that these returns are authentic; and certainly they show how very considerable a diminution has taken place in the mortality of the army" (*Times*, March 4, 1862).

The late Lord Herbert was the main agent in accomplishing this great work, which, as years pass on, will become better appreciated, more widely known, and more energetically followed up.

Professor W. T. Gairdner has happily observed, that in proportion as we are getting rid of the severer forms of epidemic disease (*e. g.*, fever, dysentery, scurvy, influenza, all more or less preventible), which had deteriorated the health of the population previously to 1848, we are also getting rid of the more severe and unmanageable types of acute inflammation. Inflammatory diseases, like fevers, he therefore justly considers to be subject to epidemic causes of increase and diminution, both as regards frequency and severity; and he believes that the acute inflammations are nearly as much under the influence of the sanitary reformer as the more obviously epidemic fevers; and, further, that some even of the chronic organic diseases have already yielded, and may be expected still further to yield, to the improved habits, the better clothing, the greater abundance of

food, and the diminished destitution of the population generally (*Clinical Medicine*, p. 42).

The Art of Medicine, guided by Sanitary Science, must now, therefore, be regarded as a productive art; for by diminishing the occurrence of preventible disease, and thereby lessening mortality, the average duration of human life has been extended to an age nearer that which has been ordained for man. Nevertheless, it behooves the Physician to remember that the sphere of his professional exertions is limited, and surrounded by insurmountable barriers; that death will eventually come alike to all, "reminding us that we ourselves must become victims to the incompetency of our art."

Dr. Pollitzer, of the Children's Hospital at Vienna, has expressed the opinion, that while the amount of mortality at early ages is diminishing in all civilized countries, under the various influences of extended hospital accommodation, care of the sick, vaccination, and general sanitary regulations, there is no corresponding increase in the strength and vigor of the human race. On the contrary, the boundaries between health and disease are becoming less and less marked. There are now to be observed numerous conditions which are undoubted deviations from the healthy standard, which it is impossible to delineate or accurately to define, because they make their appearance during a state of "*relative health*." The physician is not always even able to name the disease; and while the patient maintains that he is not feeling in health, and not looking in health, but wasting away, his food doing him no good, he has no alternative but to call himself ill. Such is the insidious mode in which many of those truly CONSTITUTIONAL diseases and DEGENERATIONS make their appearance, and which may be regarded as constituting a peculiar characteristic in the diseases of our times. These diseases are known by the various names of *anæmia*, *spanæmia*, *leucocythæmia*, *chlorosis*, to which we must also add such degenerations as those described in the previous pages, namely, "amyloid degeneration," "pigmentation," "fatty degeneration," and the ill-health (*cachexia*) of malaria, and of syphilis.

The poorness of the blood, peculiar to the class of diseases mentioned, furnishes the soil in which the feebleness and deterioration of our race is most unmistakably evident. The nervous system is, moreover, extensively involved in the diseases of the age, and thus feebleness and debility constitute their dominant character. This physical deterioration is held to be a "*sad memorial of modern civilization*." In this respect the observations of Dr. Pollitzer coincide with those of Dr. Forbes Winslow. "A constant stretch of the mental powers, a restless excitement of the passions, a perpetual struggle for advancement, the fresh wants of every day (science, and the arts themselves, being subservient even to the luxury and demoralization of the times), the destruction of all moral harmony and peace, are evils which undoubtedly prevail, and which react especially upon the younger generation." The sins of the fathers are thus being visited upon the children. The demands made upon the youth of eighteen or twenty of the present day would formerly

have been considered a sufficient tax for the strength of a man of upwards of five-and-twenty. Many of the features, also, which characterize the pathology of our age have arisen out of the treatment of infancy and childhood; and much of the deterioration of the race at large may be shown to date its origin from childhood. Thus, after seventeen years' observation in children's disorders, Dr. Pollitzer writes that *anæmia* and *chlorosis* occur alone or associated with *rickets*, *hypertrophy* of the *lymphatic glands*, and of the *spleen* and *liver*, to an incredible extent even from the first month of life. In the Children's Hospital at Vienna, from seventy to eighty per cent. he found to be thus affected. Wherever the nutrition of the child had been imperfect, the constitutional diseases associated with poverty of the blood became widely diffused. The stomach and intestinal tract first suffer, constituting the prevailing morbid condition of childhood—materially influencing the mortality at an early age, and if the age of childhood is survived, affecting the future health of youth and manhood, and doubtless of subsequent generations (*Med.-Chir. Review*, Report on Medicine, p. 261, July 1857).

The types of disease are also evidently modified by complication with other diseases, now more widely spread; and the doctrine of the incompatibility of two or more contagious diseases concurring in the same subject, has been clearly proved to be erroneous. Dr. Murchison, in an admirable paper on this subject in the *British and Foreign Medico-Chirurgical Review*, for July, 1859, has clearly shown the coexistence of *variola* and *scarlatina*; also, of *variola* and *rubeola*. Dr. F. J. Brown, of Rochester, has recorded a case of *variola* concurring with *measles* and *purpura*. The coexistence of *variola* and *roseola*, or *erysipelas*, of *variola* and *pertussis*, of *variola* and *varicella*, of *variola* and *vaccinia*, of *vaccinia* and *scarlet fever*, of *vaccinia* and *rubeola*, of *vaccinia* and *pertussis*, of *vaccinia* and *varicella*, of *rubeola* and *pertussis*, of *variola*, *rubeola*, and *pertussis*, of *scarlatina* and *rubeola*—the *rötheln* of the Germans—of *scarlatina* and *enteric* or *intestinal fever*, of *typhus* and *enteric*, *intestinal* or *typhoid fever*, have all been more or less clearly shown. Virchow relates a case of typhoid fever, combined with striking symptoms of cholera, occurring at Würzburg. Typhus fever and the marsh fevers have been observed to occur together (PRINGLE). Bilious remittents have prevailed with small-pox in the West Indies, forming, as an old writer remarks, "the most infernal combination that ever affected the human frame." Yellow fever has been associated with putrid typhus; while specific yellow fever and marsh fever, with phenomena similar in many respects to specific yellow fever, undoubtedly occur together.

There are good grounds for believing that as we approach certain well-marked geographical regions of the earth, where characteristic types of disease prevail, the confines of these disease-realms are found to mingle their types of disease together, so that the diseases of one region merge into and participate in many of the characters peculiar to the other. Cholera has extended its ravages over the earth, and is now a disease whose germs are endemic in our land;

and the system, under its influence, especially in the early cases of an epidemic, becomes rapidly depressed, to the speedy extinction of life. The *furunculoid* epidemic, which prevailed about eight years ago, was one of a novel variety. The *black death* of the fourteenth century, seems to have revived in India, and is described by the name of the *Indian Pali plague*; and it may be that the formidable disease which laid waste our country in the thirteenth century, may have arisen in these districts, and proceeded thence to our land, passing apparently in the same way that cholera has done (DR. ALLEN WEBB).

Our modern treatises on medicine justly and properly deal, largely and minutely, with the description of individual diseases, as far as their nature can be discerned; and the languages of all civilized nations attempt to define and describe them clearly. Now only are we beginning to profit by an extended inquiry into the diseases of nations; and to find that as a man wanders from his native home, the type of the diseases to which he is liable, also changes. In this field of science, an immeasurable and still unexplored country extends on every side. The more minutely, also, that individual diseases can be defined and described, the more useful will such descriptions be for comparison in future ages; and it will be seen, on comparing the descriptions of diseases in times past with accurate descriptions of the same diseases now, how, at various periods and under various circumstances, the expression of certain sets of symptoms, becomes sometimes strongly developed, while at other times, the same classes of symptoms were mild and subdued; but the really essential characters of specific diseases and morbid processes remain as persistent as the specific characters of a plant or an animal.

It will be seen how, in epidemics, many diseases have been characterized by the expression of malignant phenomena scarcely perceptible before. The small-pox nowadays is not the malignant small-pox of the time of Sydenham. Nevertheless, it is small-pox. And although it may be said that such an example does not illustrate a change in the type of a disease, because the change has been effected by artificial means; yet it must appear evident that, in effecting this change, natural results have only been imitated; and who can tell what modifying influences of a similar kind are going on, although the science of medicine can as yet take no cognizance of them? We know that certain diseases confer immunity on the individual from future attacks; may it not be, therefore, that immunity to individuals from some diseases, is conferred by agents and processes, of which we as yet know nothing; and that ultimately the types of complex morbid states may still come to be very much changed from what they are now? They appear to be changed, according to the best authorities, from what they were fifty years ago. "Many of the symptoms, and particularly the constitutional fever, usually attending internal inflammations, and resulting from cold, or from other causes independent of the application of morbid poisons, are liable to variation in like manner, although not so decidedly, nor so rapidly as the epidemic diseases,

in the course of time, and from causes not yet known. They have in fact undergone very considerable change since the early part of the present century; and it is on this account that inflammations of the lungs in particular are treated with equal success at present, with a much smaller loss of blood than they used to demand" (ALISON).

Such changes in the types of disease were formerly observed and much insisted upon by Sydenham, especially in the progress and recurrence of continued fevers; and it is now a fact well recognized, that not only does the prevalent mode of fatal termination during epidemic diseases vary, but so also do the types, peculiarities, and morbid constitutional tendencies vary in these diseases. It is chiefly with regard to the *local*, *sporadic*, or *intrinsic diseases*, and especially inflammations, such as the *cephalic*, the *pulmonic*, or the *enteric*, that any doubt exists as to whether or not they vary in their type. Distinct statements as to this fact, however, have now been made by many accurate observers, whose experience is of the utmost value to science. Dr. Alison and Dr. Bennett both agree as to the fact, "that of late years, and apparently also in different parts of the world, *inflammation*, the most important of all forms of *local diseases*, seldom shows itself with such general symptoms as demand or would justify, in the opinion of the practitioners treating them, or indeed could bear, the large bleedings which were formerly regarded as the appropriate remedy, and which accordingly are now seldom practised." There are not only also fewer examples of violent inflammation of the lungs to be met with but the *usual* (highly inflammatory) type of fever attending such inflammation has materially changed, as occurring in the present day. This change which has taken place in the *type* of the usual phenomena characteristic of inflammatory fever, cannot be explained merely by the circumstance that a previously enfeebled or diseased state of the system has brought it about in the individual. The inflammations of internal parts, such as pneumonia, now *occur often without febrile reaction*, and neither demanding nor bearing full bleedings, as described by Cullen and other authors. It is consistent, moreover, with the extensive experience of Drs. Alison, Christison, Watson, and many other physicians of the greatest eminence and long experience, that the inflammations now seldom occur with such severe symptoms of inflammatory fever as have been described at page 111, and which were the rule in the time of Cullen and of Gregory. The constitutional symptoms now attending such inflammations partake more of the type of the so-called *typhoid* state, and independent of any epidemic influence or poison having acted on the body.

The constitutional symptoms for many years past accompanying pneumonia, for instance, in this country, have been of the following kind: An enfeebled circulation; softness of the pulse, and easiness of depression by depletion, or even by taking the erect posture; tremors and feebleness of voluntary muscular motion approaching to *subultus*; indifference to surrounding objects; sickness and vomiting in some cases, with dryness of tongue and lips in others; complete anorexia or depraved appetite; in all, the symptoms gen-

erally tend to assume those of the so-called *typhoid* state rather than of *inflammatory fever*. Nevertheless, in the treatment of such cases, blood taken *sufficiently early* is distinctly beneficial; and although it may show the *buffy coat*, this has neither the thickness nor the tenacity of the *fine buffy coat* seen and described in former days; faintness supervenes on the loss of a quantity small in comparison of what was formerly well borne, and there is no such encouragement to a repetition of the bleeding, from the pulse speedily regaining its strength, or from the local symptoms abating and quickly recurring, as was formerly noticed by Cullen, Gregory, Christison, Alison, and other veterans in the practice of medicine. Dr. Gairdner also points out the great distinction to be drawn between the doctrines of Alison and of Todd in this respect. Dr. Alison recognized the disuse of bloodletting, and the increased necessity for stimulants, as a *consequence* of the changes he observed in the character of diseases. Dr. Todd on the other hand, disowned entirely the idea of such a change, and came to regard the administration of stimulants as a matter of routine. The former recognized the altered type of disease, the changes observed in its physiological manifestations, and the gradual disappearance of those forms of acute inflammation which before had appeared to require and to bear bloodletting. Dr. Todd did not recognize or acknowledge these changes (*Clinical Medicine*, 1862, p. 34).

The mode of fatal termination is also different. The tendency to death is most usually either by *coma* or by *asthenia*, as in typhus fever. The tendency to gangrene is said to have become more frequent in various inflammations of internal parts, and particularly in the lungs, within the last fifty years, than it was in the time of Cullen and of Gregory. Such are the records of experience, as stated by the late Dr. Alison in a series of papers published in *The Edinburgh Medical Journal* in 1855, 1856, and 1857; than whom the opinion of no one is more entitled to respect and consideration. "Although dead, he yet speaketh."

Dr. Handfield Jones, physician to St. Mary's Hospital, London, observes, "that instances of impaired action of the heart, sometimes amounting to serious danger, are met with at the present time with an absence of all conditions which require or justify venesection. This is associated with other symptoms of depressed nervous power, which appear to be most reasonably attributed to some kind of epidemic influence much resembling that of malarious diseases."

The type of disease which reigns at present is believed by some to have prevailed more or less since the appearance of pestilential cholera amongst us. There is now little of sthenic inflammation; bleeding and tartar emetic are but sparingly needed, while quinine, strychnine, and other tonics, with opium, and cod-liver oil, are continually in requisition in our efforts to raise and maintain failing power (*Medical Journal*, March, 1857). According to the experience of obstetric practice, the observations of Mr. Sidey, of Edinburgh, are to the same effect. He records that the epidemic of influenza in this country upwards of thirty years ago was of a most inflammatory character; cases of influenza now generally assume the *asthenic* type,

and demand a different treatment. Moreover, he observes that it is consistent with the experience of the veterinarians that the change in the type of disease has been observed among the lower animals to the same extent as in man.

In 1782, when Dr. Hamilton published his memoirs on the "*contagious catarrh*," or *influenza*, he records distinctly his belief that "our constitutions are considerably changed within the last century in Great Britain. Diseases," he remarks, "in their nature phlogistic (*e. g.*, *measles*), have appeared within the thirty years previous to 1782 less inflammatory than they formerly were, and accompanied with a considerable degree of putridity;" and bloodletting in the "*contagious catarrh*," he states with emphasis, cannot be tolerated. There seems, therefore, to have been even then an increasing belief that the degeneracy of the human race, as a whole, is in some respects advancing; and there seems some visible evidence of this more or less traceable through the past four generations.

Morel especially directs attention to the apparent increase in Europe of mental alienation, and of those abnormal states of existence which have special relations with the occurrence and existence of physical and moral degeneracy in the world; and if a comparative increase in the number of the insane cannot yet be proven, there would seem to be a tendency to more frequent complications among them of those morbid states which diminish the probability of cure, such as *general paralysis*, *epilepsy*, and a marked depression of all the intellectual and physical forces, which depression is consistent with the *asthenic* phase of present existence. *Hysteria* and *hypochondriasis*, formerly almost the exclusive appanage of the rich, the indolent, and those of a wasted life, are known to attack in great proportions the working and the agricultural classes, among whom suicidal tendencies prevail.

Dr. Forbes Winslow writes, with regard to nervous diseases, that cases of disease of the brain and nervous system are now not only of more frequent occurrence, but that a certain unfavorable type of cerebral disorder develops itself in the present age at a much earlier age than formerly. Softening of the brain, for instance, often manifests itself at the early age of thirty and thirty-five. The brain in the present day is overworked, its psychical functions are unduly exercised, strained, and taxed in the great effort required in the severe struggle and battle of life to obtain intellectual supremacy, professional emolument, and status (*Journal of Psychological Medicine*, July, 1857).

Morel again shows how imbecility, congenital or early acquired idiocy, and other more or less complete arrests of development of the body, and of the intellectual faculties, indicate the existence of children who have acquired the elements of their degeneracy during intra-uterine life. It behooves, then, all civilized governments anxiously to inquire into and to consider such facts as show,—(1.) The continued increase of suicide; (2.) The continued increase of crimes against order and law, or against the person; (3.) The monstrous precocity of young criminals; (4.) The abnormal conformations of the skull, and tendency to early union of the cranial sutures,

which prevail among criminals. So much is this the case, that in large towns, or in penal settlements and convict prisons, the physiognomy and general aspect of the "criminal population" are characteristic of a class; (5.) The general diminution of the intellectual powers, with the manifestation of the most depraved immoral tendencies; (6.) The increase in the inmates of asylums and prisons; (7.) The etiolation, blanching, anæmic or cachectic condition of the population, and any relative increase of such constitutional diseases as *anæmia*, *chlorosis*, *gout*, *Bright's disease*, *rheumatism*, *melituria*; (8.) The increased development of paralytic and convulsive affections. These are the main directions in which the degeneracy of the human race is demonstrable; so much so that in some localities the inhabitants can no longer fulfil the conditions required for military service (*Traité des Dégénérescences Physiques, Intellectuelles et Morales de l'Espèce Humaine, et des Causes qui produisent ces Variétés Maladies*. Par le Dr. B. A. Morel, de l'Asile des Aliénés de S'Yon, 1857).

The principal sources of degeneracy which appear at present to be most active in their influence for evil on large masses of mankind may be stated as follows: (1.) Degeneracy from *Toxæmia*, as from the abuse of alcoholic fluids, opium, preparations of Indian hemp (*hachisch*), tobacco, and the like; also from the effects of mineral poisons, such as lead, mercury, arsenic, phosphorus; and from the use of unwholesome vegetable food, such as diseased rye, maize, wheat, and the like. (2.) Degeneracy from the persistent and pernicious influence of *malaria*. (3.) Degeneracy from certain peculiar geological formations, soil, and water, as in the development of goitre (MACLELLAN, WATSON). See Paper on Hygiène of India, in *Med.-Chir. Review*. (4.) Degeneracy from the effects of epidemic diseases which now and then afflict large populations, profoundly influencing the system, and engendering those morbid temperaments whose types are more fully expressed in the generations which follow the one that has suffered from such epidemic pestilences. Many of such-like epidemics act like toxic agents on the nervous system. (5.) Degeneracy from the effects of the "*great town system*," as the phrase is. The chief elements of such degeneracy are (a) unhealthy situations; (b) a noxious local and general atmosphere, (c) insufficient air, (d) insufficient and improper nourishment, (e) deleterious avocations, (f) moral and social misery, wretchedness, and crime. (6.) Degeneracy from fundamental morbid states, congenital or acquired, as seen in the imperfect cerebral developments, deaf mutism, blindness, constitutional diseases, and diathesis (implanted, hereditary, or acquired), such as syphilis and scrofulosis. (7.) Degeneracy from mixed causes, from marrying in and in; and from other causes not included in the above (*Med.-Chir. Review*, Jan. 1858).

By far the most active sources of degeneracy are thus seen to be those direct and repeated influences upon the blood, the brain, and the nervous system which give rise to special morbid conditions; and which often place those who periodically expose themselves to the influence of toxic agents in a condition verging on or equivalent to insanity. The effects of *chronic alcoholism*, for instance, in giving

rise surely and progressively to the *degeneration* of the individual, are mainly demonstrable by the introduction of the following states, namely: persistent loss of appetite, indigestion, nausea; occasional diarrhœa, progressive emaciation, and cachexia; the appearance of pustular eruptions; the occurrence of eructations associated with offensive breath; serious disturbance of the function of the stomach, liver, kidneys, and heart, and the production of organic lesions in these organs, and in the structures of the bloodvessels, followed by fatal *serous effusions, dropsy, hæmorrhages, extravasations, or apoplexies*. Intercurrent with these morbid conditions, at variable periods, and otherwise contingently, "fits of drunkenness," with sexual incompetency, different forms of psychical aberration, *delirium tremens*, suicidal melancholy, and such-like morbid phenomena ensue. Finally, epileptiform seizures, general paralysis, drivelling or slavering idiocy, may close the scene. Those who become thus degenerate by alcoholic poison are arranged by Morel into two classes, namely,—*First*, Individuals who arrive at length, by a series of well-marked nervous lesions—physical and intellectual—at general paralysis. *Second*, Individuals who, although profoundly affected as regards enervation, still remain stationary at a certain state, leading a miserable existence, characterized *physically* by a special condition of ill-health (cachexia and marasmus), and *morally* by manifestation of the worst tendencies, and of the lowest animal propensities. The serious degenerative effects thus detailed in their extreme forms, resulting from the poison of alcohol, ultimately influence the procreative functions; first, by diminishing the vital standard of the offspring; and second, by annihilating the generative power altogether. When such results are coupled with the moral and social aberrations which ensue from bad example, misery, and want, in families and masses of men, they become ample sources of the degeneracy and degradation of the population, not only throughout the existing but succeeding generation; and not only is the vice of alcoholic abuse hereditarily transmissible (MOREL,) but it also frequently leads to insanity in the offspring of the drunkard (WHITEHEAD, ADAMS); and in cases where the tendency to alcoholic excesses has an hereditary origin, the cure of the dipsomaniac is generally impossible. Morel gives the following example, in which a well-marked succession of morbid phenomena became developed in different descendants of a family throughout four generations. The great-grandfather of the family was a dipsomaniac; and so complete was the transmission of the disease, that the race became totally extinct, under the well-marked phenomena of alcoholic poisoning and degeneracy. The effects entailed were: in the first generation, alcoholic excesses, immorality, depravity, brutish disposition; in the second generation, hereditary drunkenness, attacks of mania, general paralysis; in the third generation, sobriety prevailed, but hypochondriasis, lypomania, persistent ideas of persecution, and homicidal tendencies were expressed; in the fourth generation, intelligence was but feeble, mania became developed at sixteen years of age, stupidity running on to idiocy, and to a condition involving extinction of the race. Sir James Clark also made the observation, more than twenty

years ago, that the constitutions of the past three generations had deteriorated progressively from father to son (*Treatise on Pulmonary Consumption*, p. 11).

The persistent pernicious influence of the marsh poison will be fully noticed in the last part of this treatise; but there are other abundant sources of constitutionally morbid influences; and, whatever the explanation may be, it certainly is a matter of fact, as the late Dr. Alison wrote, that the *constitutional affections*, going along with the same extent of inflammation and its local effects, are extremely various in different persons previously alike in good health, or even in the same person at different times; and that we are not entitled to deny that what happens in this way in different individual cases may not happen also in nations and in seasons. As we are still very imperfectly informed as to the mode in which any local inflammation excites constitutional fever, we have no reason to doubt that the constitutional reaction consequent on the excitement of a certain degree of inflammation of the lungs may vary, equally as that which is consequent on the introduction of a certain quantity of the poison exciting *typhus fever*, *measles*, *scarlatina*, or *cholera*; in all of which the previous muscular strength goes for nothing in determining the degree or danger of depression or debility which may ensue.

Dr. Christison has recently communicated to the Medico-Chirurgical Society of Edinburgh his experience relative to the changes which have taken place in the constitution of fevers and inflammations in Edinburgh during the forty years previous to 1857. His experience, as well as that of many of the older physicians of Edinburgh, shows that a transition had and did every now and then take place from an *inflammatory form* of fever to one of an *asthenic type*; that it was necessary, on the outburst of any epidemic, to watch carefully the early cases, to observe the mode in which the fatal cases terminate, and to observe generally the *constitutional tendency* or type of individual cases, in order to form an accurate judgment of the general character of the epidemic about to prevail. It will be seen also, on referring to the most approved and recent works on the diseases of India, that the descriptions of inflammations as well as fevers now seen there, when compared with the statements of Dr. Johnson, Mr. Twining, and others, twenty-five or thirty years ago, may be held to indicate that there has been a change in the usual form of *reaction* in inflammatory diseases in that climate as well as here. Such conclusions may be inferred from the experience of Dr. Morehead, recorded in his *Clinical Researches on the Diseases of India* (vol. ii, pp. 71, 72, and 359); and the experience of Sir Ranald Martin, expressed in his recent classic work on Climate, bears out the same observation: consult also Graves's *Clinical Medicine*, vol. i, p. 303, for the doctrines of Autenrith and Graves on the change of type in disease; and for evidence regarding the deterioration of race, see p. 105 of Mr. Simons's *Third Report on the Health of Towns*.

It appears evident, therefore, that the *human body* is capable, from causes known as well as unknown to us, of undergoing various al-

terations as regards not only its *physical*, but also what has been termed its "*Medical Constitution*." *Fevers* are known to change their types; *epidemics* to assume new tendencies; and *inflammations* and *local lesions* to affect in no constant manner the constitution of individuals at the same period, or at different times and in different countries. This view of the subject may be summed up in the eloquent language of Dr. Watson, when he writes,—“I am firmly persuaded, by my own observation, and by the records of medicine, that there are waves of time through which the *sthenic* and the *asthenic* characters of disease prevail in succession, and that we are at present living in one of its adynamic phases” (*Edinburgh Monthly Journal*, June, 1857). It must be admitted, however, that much of what is written and here quoted regarding changes of type is based on the uncertain impressions of individual men, sometimes handed down by tradition, rather than based on the results of a deliberate deductive inquiry from statistics applied to the question by men agreed as to the meaning of terms. On the other hand, I think Dr. Markham, in his very able Gulstonian Lectures on “The Uses of Bloodletting in Disease,” puts the evidence regarding change of type in disease on much too narrow a basis—not to say an erroneous one—when he holds that it is merely an excuse put forward for a change of practice in inflammations, and that the only argument in favor of such a theory rests upon the assumed conclusion that venesection produces different effects now from what it formerly did.

A somewhat similar view of this important subject has been ably advocated by Dr. J. H. Bennett, of Edinburgh. He contends that inflammation is the same now as it has ever been—that the analogy sought to be established between it and the varying types of fevers is fallacious—that we cannot place reliance on the recorded experience of the past—and that our recent changes in therapeutics are solely due to an advanced knowledge of diagnosis and pathology (*Principles and Practice of Medicine*, p. 267).

The local phenomena of inflammation are undoubtedly constant; but the question of change of type has reference to the constitution of the individual, and to the constitutional modes by which the inflammatory state is expressed in a given number of persons. Much of the argument of Drs. Markham and Bennett appears to me to be beside the question; and there are certainly good grounds for believing that elements of constitutional degeneration exist in such abundance, especially in the communities of large towns, that Dr. Watson has, I think, happily expressed the consequence of such deterioration in the sentence quoted from his writings on the subject.

CHAPTER X.

MODES BY WHICH DISEASES TERMINATE FATALLY.

OUR knowledge on this subject is derived chiefly from three sources, namely, from the examples and illustrations afforded by the study of—(1.) Death from old age; (2.) Death from fatal injuries; (3.) The powers and actions of all our best remedies. Such study leads to the important practical conclusion, that the same lesions of important organs may prove fatal in very different ways, and the fatal event may be averted by very different and very opposite remedies (ALISON). It is also to be observed, that in constitutions which are unimpaired, and, indeed, in every morbid process, there may be recognized a tendency to a spontaneous favorable termination.

Death happens either from the decay of life, as in old age; or it happens as an accident caused by some of those untoward lesions or derangements of the vital organs which occur in the course of the various diseases and injuries to which mankind are liable.

Death by *extreme old age*, may be considered in many instances, as the desirable end of a long-continued and perhaps a dreary journey. The sufferer appears to fall asleep, as he might do after severe fatigue; and the long and weary journey is thus often brought to a close with little apparent derangement of the ordinary mental powers. The final scene is often brief, and the phenomena of dying are almost imperceptible. The senses fail, as if sleep were about to supervene; the perceptions become gradually more and more obtuse, and by degrees the aged man seems to pass into his final slumber. We scarce can tell the precise instant at which the solemn change from life to death has been completed. Sensation fails first, then voluntary motion; but the powers of involuntary muscular contraction, under the excitement of some external stimulus, may continue for some time longer to be feebly expressed. The blood ceases at first to be propelled to the extremities. The pulsations of the heart become less and less efficient. The blood fails to complete its circuit, so that the feet and hands become cold as the blood leaves them, and the decline of temperature gradually advances to the central parts. Thus far the act of dying seems to be as painless as that of falling asleep; and those who have recovered after apparent death from drowning, and after sensation has been totally lost, assert that they have experienced no pain. What is called significantly the *agony of death*, may therefore be presumed to be purely automatic, and therefore unfelt. The mind, doubtless, at that solemn moment, may be absorbed with that instantaneous review of impressions made upon the brain in bygone times, and which are said to present themselves with such overwhelming power, vividness, and force, that, in the words of Montaigne, "we

appear to lose, with little anxiety, the consciousness of light and of ourselves." At such a time, the vivid impressions of a life well-spent, must constitute that *euthanasia*—that happy death—to be desired by all.

The untoward lesions or derangements of vital organs, which occur during the progress of disease, terminate the life of man by various modes of dying. While it is ordained that eventually all must die, yet it is possible sometimes to avert, for a time, the tendency to death. To know by what agents this may be properly accomplished, it is necessary to know the modes in which death may approach in disease. Dr. Watson has happily observed that life rests upon a tripod, whose three vital supports are, the *heart*, the *brain*, and the *lungs*. Through the impaired functions of some one or more of these organs, the tendency to death is expressed. The mode of dying may begin at the *head*, the *heart*, or the *lungs* (BICHAT). But inasmuch as the functions of these organs are mutually dependent upon each other, so impairment of function in any one of them may ultimately lead to death, while the mode of dying is expressed chiefly through the functions of another. The mode of dying in disease is usually a complex one, for many parts thus mutually dependent on each other, are more or less immediately involved. Therefore it is of the greatest practical importance to observe how and when the different functions begin to languish, and how they may be best sustained in their exertions to maintain life.

When a person loses blood to such an extent that he faints, as from a wound, or by hemorrhage occurring in disease, and if the flow of blood is not arrested, the state of *faint* or *syncope* continues, is not recovered from, and the heart's action ceases; not because it is unable to contract, but because its natural stimulus, the blood, is withdrawn from it, or does not arrive in sufficient quantity to be of use. This is called death by *anæmia*. In such cases, if blood can be timeously supplied to the heart (as by the operation of transfusion from a healthy person into the patient who is losing blood), the suspended function of the heart may be restored, and a supply of blood, sufficient to maintain life for a time, may be thus obtained.

The symptoms of approaching death by this mode of dying are, paleness of the countenance and lips, cold sweats, dimness of vision, dilated pupils, vertigo, a slow, weak, irregular pulse, and speedy insensibility. If the hemorrhage has been sudden, in large quantity, as from the uterus in "flooding," there may be nausea, or even vomiting, restlessness, tossing of the limbs, irregular sighing breathing (*anxietas*), delirium, and one or two convulsions before death ensues.

But another mode of death may be more immediately connected with the heart itself, and be independent of the supply of blood. In other words, the stimulus from blood may be sufficient, but the contractile power of the organ may fail. Such a mode of death is by *asthenia*. Many poisons act in this way, and many diseases which are due to morbid poisons in the blood tend to prove fatal

by this mode of dying. Cases of extensive mortification of parts, of acute inflammation of the peritoneum, and of malignant cholera, die in this way.

The symptoms consist in the pulse becoming feeble and frequent, and ultimately failing altogether to be perceived. The muscular debility becomes extreme, but the senses remain perfect, often painfully acute, and the intellect clear to the last.

Persons whose death is by *anæmia* or by *asthenia* are often spoken of as having died in a faint, or by *syncope*; and there is still a mode of dying intermediate between the two, the type of which is seen in *death by starvation*.

Death may also be produced by the suspension of the functions of respiration, as when access of air to the lungs is prevented by a direct obstruction, either to the air-passage, in choking, or to the action of the chest, so as to prevent its expansion; or when the actions of the muscles of respiration cease, in consequence of disease or injury to the brain producing insensibility. The first of these modes by which the respiratory functions are suspended is that known as *suffocation*, technically expressed by the term *apnœa*, or privation of breath. Examples of this mode of dying may be referred to in cases of *drowning*, *smothering*, *choking*, *strangulation*, *throttling*, and closure of the rima glottidis by foreign bodies. In Dr. Allen Thomson's anatomical museum at Glasgow there is a larynx preserved in which a piece of coal is wedged between the rima glottidis. A collier thus died by *apnœa* or *suffocation* produced by the piece of coal dropping into the larynx while he lay on his back in the mine, excavating the coal from the roof of the coal pit. Forceful pressure upon the chest, as sometimes happens in crowds during a continued crush of people, or occurs to workmen who have been buried by falls of earth and rubbish; in short, whatever causes an immovable condition of the lung-case beyond a period of three minutes and a half will thus produce a fatal result. Tetanus, and the influence of strychnine, prove fatal in this way. Morbid states produced by disease, and which terminate fatally by *apnœa*, are *œdema* of the glottis; disease of the spinal cord above the origin of the respiratory nerves (phrenic, intercostals, and spinal accessory); effusion of serum into the pleural cavities; sudden infiltration of the lungs by inflammatory exudation, or collapse of the lung in bronchitis.

The symptoms of approaching dissolution by this mode of dying are, strong but ineffectual efforts to contract the respiratory muscles, and struggling efforts to respire, amounting to agony, of short duration, followed by vertigo, loss of consciousness, and convulsions; at last all effort ceases, twitchings or tremors of the limbs alone remain, the muscles relax, and the sphincters yield. The heart and the pulse, however, still continue to act after all other signs of life are past. The recent experiments instituted by the Medico-Chirurgical Society of London show that, on an average, the heart's action continues for three minutes fifteen seconds after the animal has ceased to make respiratory efforts. On this last circumstance rests our hope of resuscitating persons so suffocated, if artificial

respiration be timeously resorted to, and persevered in. This prolonged action of the heart circulates blood, which is dark, venous, and not arterialized, and accordingly the face, at first flushed, becomes turgid, and then assumes a livid and purple hue; the veins of the head and neck swell, and the eyeballs protrude from their sockets. At length the heart ceases to beat, and life is extinct (WATSON).

Death by coma occurs when there is a loss of consciousness first, with the appearance of profound sleep, from which the patient may be partially roused. The symptoms of approaching death by this mode of dying consist in a gradual blunting of sensibility to outward impressions, slowness of respiration, the inspiratory effort being often delayed, and then performed with a sudden noise and jerking inspiratory effort, technically known as *stertorous* breathing. All voluntary attention to the act of breathing is lost, but the influence of a reflex stimulus to its performance continues. At length this function fails also. The chest ceases to expand, the blood is no longer aerated, and thenceforward precisely the same changes occur as in death by apnoea.

Such are the several modes by which death tends to approach; and "*to obviate the tendency to death*" is a doctrine which was often and strenuously inculcated by Cullen. After him, no less earnestly has it been impressed on many by my respected teacher, the late Professor Alison, whose interesting Lectures on *fevers* and *inflammation* furnished numerous illustrations. To his *Outlines of Pathology and Practice of Medicine*, and to the first volume of Dr. Watson's *Lectures on the Principles and Practice of Physic*, the student is referred who would seek further information—sources whence the preceding observations have been mainly compiled relative to the modes by which death may approach:

"Many are the ways that lead
To his grim cave, all dismal; yet to sense
More terrible at the entrance than within."

"It is as natural to die as to be born;"

and thus, in

"Passing thro' Nature to Eternity,
The sense of death is most in apprehension."

CHAPTER XI.

PRINCIPLES WHICH DICTATE THE TREATMENT OF THE COMPLEX MORBID PROCESSES.

I. *As regards Fevers or the Febrile State.*

To avert the tendency to death in the febrile state, it is necessary to observe how fevers naturally terminate favorably. Four modes are enumerated by Dr. Parkes, namely:

1. *By crisis*, in which the temperature falls suddenly in a few hours, and usually with some abundant excretory discharge, in which, possibly, much of the water which has been retained in the system is poured out.

2. *By lysis*, in which the fall of temperature is gradual from day to day, till the normal standard is attained. The decline may thus occupy many days, the thermometer being known to take seven days in falling from 102° to 98° Fahr.

3. *By a combination of these two modes*, namely, by a sudden fall of temperature to a certain point, and then a gradual decrease to the normal heat.

4. Another mode has been observed by Dr. Parkes, namely,—*By a somewhat irregular alternation of febrile and non-febrile periods, as shown by the temperature and the issue.*

When *fever* terminates by any of these modes, convalescence commences, normal nutrition is renewed, and the body begins to gain in weight. The blood is poor in albumen and in red particles; and there is now a danger that the rapidity of metamorphoses of tissue will exceed the healthy standard, as shown by the great tendency which convalescents from fever have to lose heat. The temperature may fall, and the excretions may diminish below their healthy amount. Great care, constant attendance, and watchfulness, are required when the patient begins to convalesce, if the fever has been long and severe; and the treatment of the febrile state itself may be thus generally stated as consisting in a combination of measures,—(1.) To reduce excessive heat; (2.) To insure sufficient but not excessive excretion and elimination of the excretions; (3.) To act on the exhausted and semi-paralyzed nerves; (4.) To neutralize any specific poison which may have set up the fever, and so to improve the state of the blood; (5.) To relieve distressing symptoms; and lastly, To obviate and counteract local complications (PARKES, MURCHISON).

To accomplish the first indication, Dr. Robert Jackson, "the patriarch of Military Medicine," and after him Dr. Currie, of Liverpool, practised to an extreme degree the application of cold water. In health such an application tends to increase the metamorphoses of tissue, as shown by Lehmann and Sanderson; and therefore its excessive use is contraindicated in the febrile state. Bloodletting or hemorrhage also tends to reduce temperature; but bloodletting can rarely be tolerated in many specific fevers, such as typhus, typhoid, scarlatina, and the like. *Infusion of digitalis* has been found by Wunderlich to have a wonderful influence in reducing and moderating the temperature in many febrile states, such as typhoid fever. Its most obvious action in small doses is to depress the force of the heart. The dose should therefore be cautiously regulated: it must not be repeated too soon, nor be increased, if it should not operate at once.

To insure sufficient but not excessive excretion, and to promote its elimination in fever, is much more difficult than to reduce temperature; which, for obvious reasons, it is not always judicious to attempt.

The system ought to be supplied with an abundance of alkaline salts, if the urinary excretions are not eliminated. *Chloride of sodium, the alkaline salts of soda and of potash*, tend to aid the formation of urea and its elimination. Purgatives generally, and especially *salines*—i. e., salts of the alkaline and earthy metals—tend to insure a proper excretion, probably by removing from the blood some of the abnormal products formed in fever, and great relief may follow their moderate use. Where urea is retained they promote its elimination, because it is known that urea sometimes passes off by the mucous membrane of the intestines. But in some fevers, as in typhus, purgatives must be very cautiously and sparingly given, and always in mild doses. So also elimination by the skin, to the extent of *diaphoresis*, is to be dreaded in typhus fever. (See “Treatment of Typhus Fever.”)

The most important indication, however, in the management of the febrile state is to find some substance which, being “restorative” in its action (HEADLAND), will so act upon the blood, and thus restore the exhausted energies of the nervous centres. Food, mild stimulants, and quinine are all more or less employed; and quinine especially may be employed with benefit. Infusion of coffee as a medicine has been given by Dr. Parkes, with the beneficial effect of relieving headache. Böcker and Lehmann have shown that the use of coffee, in health, delays the metamorphoses of tissue, and excites the nervous system. The special treatment of the febrile state depends on the diseases of which it forms a part, and by which it is more or less modified—forming a special topic for consideration in the part which treats of special diseases. But it is above all necessary to guard against the habit of trying always to be doing *something*. As a routine system, nothing can be laid down as a rule, either in the direction of depletion, or of evacuants, or of stimulation. The febrile state is in many diseases part of the essence of the morbid state, which cannot be cut short nor materially subdued by remedies. There is no specific remedy for the cure of any fever; and in the present state of our knowledge regarding specific febrile diseases, there can be no specific remedy for their cure.

Every disease where fever is present, and every case of specific febrile disease, must be studied, so that its management or treatment may be regulated on the merits of the individual case; and must be regulated by the state of each particular function as determined by clinical investigation daily.

II. As regards Inflammation.

It is necessary clearly to understand and to bear in mind that, in the first instance, it is not the *lesion* which may attend the inflammatory process as a result, which is to be attended to; but it is the *diseased action tending to the lesion* which it is the object of the physician to overcome, to subdue, and turn aside; and that the occurrence of any lesion is, if possible, to be prevented. It is to the *strictly vital action—the excitement of tissue*—which tends to the organic lesion, that remedies must be applied, in order to avert the

tendency to lesion, expressed by the symptoms of a constitutional kind, already referred to.

The treatment which will subdue this tendency has been technically called "*antiphlogistic treatment*." Its mode of action depends upon the regulation and adoption of every agent, plan, or circumstance most favorable to the subsidence of the inflammation, and which will favor the influence of remedies, and oppose the advance and persistence of the inflammatory state. The treatment embraces,—(1.) Antiphlogistic regimen; (2.) Antiphlogistic remedies—means for reducing the quantity and altering the quality of the blood, and thus to counteract in a direct way, the complex morbid phenomena of inflammation.

The *regimen* consists in,—(1.) A sparing allowance of non-nutritious diet; the administration of bland, simple, and cooling drinks, given often, and in small quantities; (2.) Absolute rest of body and mind; (3.) Residence in a well-ventilated apartment, maintained at an equal temperature, day and night, of about 62° Fahr.; and in cases where the lungs are inflamed, a temperature of not less than 70° Fahr. to 80° Fahr., and as dry and pure a state of the atmosphere as possible (VEALE).

The *remedies* comprehend *bloodletting*, *purgatives*, *antimonials*, *mercurials*, *opium*, *alkalies*, and *saline drugs*.

The most important and the most efficient of these remedies is undoubtedly *bloodletting*; while it must at the same time be remembered that it is not every case of inflammation that requires or warrants the abstraction of blood. It is a spoliative remedy, powerful for good and for evil. In the treatment of inflammation, it has been well observed by Dr. Watson that "each case requires its special study, speaks its proper language, furnishes its peculiar indications, and reads its own lessons." The carefully recorded facts of well-conducted though empirical observation, for hundreds of years, have attested the immediate sanative influence of bloodletting in *incipient* inflammation; and the most eminent physicians of bygone modern times have recorded, in unmistakable language, how potent is this remedy for good, and the reasons for their belief.

Our forefathers well knew when the body suffered from an inflammation in the "*inward parts*;" and in saying this we give them credit for far less scientific knowledge than they really possessed. Against such inflammations, whether in the *head*, the *chest*, or the *abdomen*, they learned, by "*watching* and not by *counting*," the sanative efficacy of early venesection; and they obtained most trustworthy evidence and experience of its power to control inflammation. Following up such doctrines will be found those veteran physicians who hold the foremost rank in the science of medicine of the present day in this country. The doctrine generally taught and universally acted upon with reference to bloodletting in inflammations is, "*so to bleed as to secure the advantages of the remedy, and to avoid its disadvantages*" (WATSON).

The standard examples of what bloodletting can do soon become apparent to every surgeon's apprentice, or hospital pupil, if he does not himself swoon the first time he sees the blood flow from the

patient whom his master and teacher may wish to relieve. He may see the apoplectic sufferer roused to consciousness while the blood yet flows from the vein; and he may observe, also, that the *stounding pains* of the head in cephalic inflammations are immediately relieved, that the impatience of light and sound, the frequent, sharp, intermittent pulse, with vomiting or nausea on assuming the erect posture, the tendency to squint,—in short, all the urgent symptoms of incipient encephalitis, at once, or one by one, disappear as the blood continues to flow. He may also notice in *thoracic* inflammation that the pain, the dyspnoea, the tightness of the chest, all disappear. Dr. Alison, as regards pleurisy, and Dr. Watson, as regards inflammation of the bowels, bear personal testimony to the good effects of bloodletting. They experienced its sanative influence in their own persons, and the practice undoubtedly saved their valuable lives from these respective diseases. Testimony from such personal experience has also been borne by the late Dr. Gregory, of Edinburgh, and before him by the celebrated Dr. Radcliffe; and so also is the testimony of many who, having experienced the benefit of the remedy once, imagine that, when again attacked with inflammation, they may be again relieved by its use. Of any one of these illustrious examples from personal experience it might be said, as Dr. Gregory said of Dr. Radcliffe, that “he was at least no fool; and we may depend upon it he would not have allowed a hundred ounces of blood to be taken from him in one day without good reason for it” (*Edin. Med. Journal*, March, 1857).

“Although much has been done,” writes Dr. Alison, “particularly by the French pathologists, to enable us to judge of the texture within the chest which is the subject of inflammation, and although this is a matter of real importance, because we know that the history of the changes to be expected from inflammation in the bronchiæ, substance of the lungs, and pleura, is materially different, and of course the diagnosis of these gives us great advantage in studying the progress of any individual case,—yet as to the specific questions of bloodletting or not, the quantity, or the repetition of the bloodletting, our predecessors were very nearly as well informed as we are. It is an important practical error,” he also continues, “to fix the attention, particularly of students of the profession, too much on those characters of disease which are drawn from changes of structure already affected, and to trust too exclusively to these as the diagnostics of different diseases; because, in many instances, these characters are not clearly perceptible until the latest and least remediable stage of diseases. The very object of the most important practice, moreover, in many cases, is to prevent the occurrence of the changes on which these lesions depend. After these lesions are once established, the cases are very often hopeless, or admit only of palliative treatment. In those diseases in which most can be done by art, our practice must always be guided in part by conjecture, because, if we wait for certainty, we very often wait until the time for successful practice is past; and therefore, although an accurate knowledge of the whole history of each disease is essential to its proper treatment, yet, in a practical view, the most important part of its history is the assemblage and succession of symptoms, by which its nature, at least, if not its precise seat, may often be known, before any decided lesion of structure has occurred. Accordingly, when

this department of pathology is too exclusively cultivated, the attention of students is often found to be fixed on the lesions to be expected after death, more than on the power and application of remedies, either to control the diseased actions or relieve the symptoms during life."

The immediate effects of loss of blood as a remedy in inflammation, are,—(1.) A sedative result on the heart's action, by diminishing the pressure on the vessels, by diminishing the quantity and altering the quality of the blood. The withdrawal of a considerable amount of stimulus from the central organ has thus a depressing effect, greater in proportion to the sudden loss of blood—the excitability of the nervous system being thus also reduced. (2.) The loss of blood generally has a derivative influence upon the blood in the part about to become the seat of effusion. This has been seen to occur in experiments upon the transparent parts of animals. (3.) Bloodletting facilitates the action of other remedies. (4.) While the effect of bloodletting is well known, by the observations of Louis and Alison, not always to check the extension of the sphere of inflammation, *yet it essentially modifies* its character, (a) sometimes by limitation of its sphere to a certain extent, (b) by diminishing the quantity of blood from which the inflamed part is nourished, (c) rendering the fibrinous exudation more liable to reabsorption.

Of late, the medical profession has proclaimed with no uncertain sound, especially from the metropolis of Scotland, as to the good effects of bloodletting in the treatment of inflammation. It is well known that no remedy demands a more careful study of its application, or a more delicate adjustment of its powers; and therefore some general rules may be here stated as a guide in the use of the lancet.

1. The utility of bloodletting varies with the variations in the type of disease. A change in the type of inflammatory diseases (*i. e.*, in their usual symptoms, local and general, in their tendencies to certain local and general results rather than others, or a change in their mode of tending to a fatal termination) demands a new study and fresh adjustment of the remedy in each particular case, country, locality, and epidemic.

2. There is no truth, perhaps, in medicine more conclusively determined than that we ought not to bleed, or, if we do so, we must bleed sparingly, when the inflammation depends on or is associated with the action of a morbid poison. In epidemics, therefore, of every kind, we should not hastily have recourse to the lancet, but should remember that the disease probably depends on a poison, has a course to run, and is not amenable to the mere abstraction of blood.

3. It is necessary to observe carefully, and to watch the combination and succession of the constitutional and local symptoms from the commencement of the febrile attack, and so to judge as to the propriety of bloodletting.

4. In order to obtain the good effects of the remedy by a full bleeding, it must be done *prior* to fibrinous effusion or new growth;

seeing that it is *the diseased action which tends towards the lesion*—namely, effusion or growth of new material—which the physician desires to control by this remedy.

5. When the symptoms of *inflammatory fever* are little complicated and seen early, in persons previously healthy, the more violent the symptoms are, the more intense and rapid the constitutional reaction, if it does not indicate exhaustion; and the more decided the change on the function of the part affected, the more confidently we may depend on the effect of full bloodletting in relieving them.

6. When the symptoms of inflammatory fever have been uncertain and insidious in the beginning, so that the early stage has passed over unchecked, or modified by previously existing constitutional disease, or complicated with organic local disease, or when they denote debility, exhaustion, or the so-called typhoid state, they generally prove improper cases for bloodletting, even when seen within the first few days.

7. Generally, it may be stated, that when the fever is high (above 104° Fahr.), when we may be sure that over a part of the inflamed organ there is congestion, stagnation of blood, distension of vessels, commencing extravasation, and change of the constitution of the blood—but these latter changes still partial and not far advanced—the power of bloodletting to control the disease has been clearly established.

8. The nature of the membrane or organ affected must always be considered in estimating the propriety of bleeding. If a serous membrane, for instance, be actually inflamed, the patient, for the most part, bears bleeding well, and is usually greatly relieved by it. With respect to organs, it is found that inflammation of the brain is less influenced by bleeding than inflammation of the liver, and inflammation of the liver than inflammation of the lungs. The gist of the evidence brought forward in Dr. Markham's very able and interesting Gulstonian Lectures establishes the special efficacy of venesection in those inflammations which are accompanied with obstructions of the cardiac and pulmonary functions, but do not prove it to be useless nor injurious in the cure of acute inflammations generally, if its remedial functions are properly used, and the advantage gained be duly followed up by appropriate remedies, such as are to be presently noticed. The symptoms which demand a full bloodletting in pneumonia are also those which indicate the greatest danger—namely, violent pyrexia, usually beginning suddenly, with full, strong, hard, and quick pulse—urgent *dyspnœa*, even *orthopnœa*—swelling and flushing of the face, frequency and violence of cough, with scanty or truly pneumonic expectoration, aggravating the pain which extends through the chest,—when such symptoms are seen within *three* days of their commencement, especially in those of robust and full habit in the prime of life, bloodletting is the remedy to be used,—everything else is *trifling*, and it is *not safe* to dispense with it. The nearer a case answers this description, the more sure we may be that the effect of bloodletting will be satisfactory, and its repetition, if the symptoms shall recur, will be well borne (ALISON).

But if the inflammation be so limited that it produces little or no appreciable impediment to the respiratory or cardiac functions, when the temperature does not exceed 104° Fahr., when the pulse does not exceed 120, and when the respirations do not exceed 40 in the minute, such cases of pneumonia will get well by antiphlogistic regimen alone.

9. It is found in practice, also, that this most powerful of therapeutic agents in the cure of inflammation requires the greatest caution in its repetition, for there is a line beyond which bleeding becomes destructive instead of remedial. Two indications are of great use in determining as to the propriety of a second bloodletting, namely,—

(a.) *As to how the first bleeding is borne*—a test first suggested by the late Dr. Marshall Hall. If much blood flow from an ordinary-sized opening, before any tendency to syncope manifests itself, venesection is then considered to be well borne; if, on the contrary, the patient soon faints after a vein is opened, the judicious practitioner desists from further depletion. The urgent symptoms, being thus relieved for the moment, may again return, after a longer or shorter interval, and thus demand a repetition of the remedy, to be now judged of (b) by the reaction of the system generally, as indicated by the state of the local symptoms, their urgency for relief, the character of the pulse, and the appearance of the blood first drawn. The reaction may be of such a kind that a *sthenic* state of *inflammatory fever* still continues, or returns after temporary subsidence. The inflammatory process having been interrupted, so far modified, but not arrested, the remission proves transient, and the reaccession may be more fierce than the onset. A repetition of bloodletting is demanded so soon as such reaction has declared itself. On the contrary, the reaction may be *asthenic*, or of nervous character, the pulse being rapid, soft, and jerking, the breathing oppressed, headache and *tinnitus aurium* present, with general nervous excitement; bleeding, under such circumstances, *is not to be repeated*. A full opiate will allay the nervous excitement.

10. The next consideration is, "What indications for bleeding are to be drawn from the state of the blood?" The blood offers certain indications, either for bleeding or refraining from it, when the symptoms would otherwise demand or forbid this operation. The firmness of the coagulum, for example, has been considered at all times as a mark of the tonic state of the system, and as a warranty for repeating the bleeding when the part is as yet unrelieved, and the reaction continues of the *sthenic* type.

The thickness, and especially the firmness, of the *buffy* coat, if lifted on a pin, was one of the leading characteristics of the existence of acute inflammation, amongst others already noticed, and was much founded upon by Dr. Gregory, as guiding his practice in the treatment of inflammation. On the contrary, a looseness of texture of the clot is a sure sign of great debility, so that, unless other circumstances strongly indicate the necessity of bleeding, it ought not to be repeated when the yellow or buffy substance on the surface is loose and not thick.

11. The proportion of the serum to the clot, and also its occasionally altered characters, are arguments for or against bleeding. When the quantity of serum is unusually large, unless the clot be very firm, bleeding ought not to be repeated. Also, when the properties of the serum are so altered that it coagulates and forms one mass with the clot, bleeding is constantly prejudicial; and, lastly, it has been observed that when the serum, which has little or no affinity for the red globules in health, readily dissolves them, it is an unerring sign that further bleeding should be avoided. In some of the febrile diseases the fibrine never augments, remains often in normal quantity, and is also often diminished. In the acute inflammatory fever, on the contrary, there is a constant augmentation of this principle, compared with the red globules, as observed by Andral. It is this excess of fibrine which gives firmness to the clot, and is the cause of its being "buffed" and "cupped." The immediate effect of bleeding, according to the same high authority, is to reduce the red globules, but not so with the fibrine; for a reduction of the fibrine does not take place till after a certain time. Such is the state of the blood in the sthenic inflammatory states. There are many reasons, however, for not esteeming the buffed and cupped state of the blood, denoting an excess of fibrine, as a sufficient warranty for bleeding; for these conditions are often present in erysipelas, phthisis, or the early stages of typhus fever; and in either of these cases the loss of a moderate quantity of blood might hurry the patient to his tomb. Again, in acute rheumatism the blood is not only buffed and cupped, but contains a maximum quantity of fibrine; yet the best practitioners seldom think it necessary to take blood, considering that mode of treatment as neither affording present relief nor shortening the course of the disease. The fact, then, of the blood being buffed and cupped does not in all cases warrant venesection. It is also well known that the sthenic or buffed characters of the blood are often greatly modified by the manner in which the blood is drawn; thus, if an individual be bled in both arms, and the blood allowed to flow with different velocities—that is, in a full stream from one and slowly from the other—the blood drawn is identically the same; yet a thick buff will be wanting in the latter and be present in the former. And if the apertures be of different sizes, the same differences will result; the blood from the larger orifice will be buffed, while no such effect is seen in the blood drawn from the smaller one. Again, the form of the vessel which receives the blood, as to whether it be flat or conical, and also its temperature, or whether the blood be received into one that is cold or warm, will also affect the phenomena of its coagulation.

There are many circumstances, therefore, which prevent the blood from being an unerring guide for bleeding in cases of inflammation; but *the assemblage and succession of symptoms* must decide as to the propriety of bloodletting in doubtful cases.

12. An improvement in the character of the secretion or excretion from the inflamed part contraindicates the repetition of bloodletting; for instance, in pneumonia, if the *character* of the expectoration, from being scanty, tenacious, and tinged with blood, becomes

copious and free, much may be expected from this natural tendency to cure.

13. It is an object to effect the sanative result with as little expenditure of blood as possible; but the amount to be taken can only be judged of by the effects produced. The patient should be bled, if possible, in the upright position, and a full stream of blood allowed to flow from a sufficiently large orifice in a vein. To accomplish this fully, it may sometimes be necessary to open a vein in each arm, so that the flow may be from both at the same time.

Bloodletting may be employed either generally or locally. General bleeding is best adapted to subdue acute inflammation of visceral organs, because it makes a more decided and rapid impression upon the system. Local bloodletting, by leeching, scarification, or cupping, is more useful in chronic inflammation, and in inflammations affecting membranes, such as the pleura, peritoneum, meninges, and articular membranes; but it is often advantageous to combine the two methods of taking blood. In the case of external visible inflammation, the direct abstraction of blood from the inflamed part during the congestive period of the inflammatory process, is seen to exercise a benign influence over the progress of the inflammation.

The next most important class of antiphlogistic agents in the treatment of inflammation consists of *purgatives*, especially the *resinous cathartics*, like *scammony* and *gamboge*. (1.) They free the stomach and intestines from accumulated food and feces, or other irritating and acrid matters. (2.) They subdue the inflammatory tendency by the discharge of a large quantity of serous fluid charged with albumen, from a large extent of mucous membrane. Thus they tend to check effusions, and diminish the force of the heart's action. Their use is especially indicated in *encephalic* inflammations and *hepatic* congestions; but they are less efficient in subduing *thoracic* inflammation; while in the *enteric* inflammations they ought not to be pushed beyond merely unloading the alimentary canal. Combined with diaphoretics, they promote the elimination of morbid materials through the glands.

The influence of mercury varies with its mode of administration and the constitution of the patient. It is followed, in large doses, by an increased flow of watery evacuations from the bowels, and an increased flow of saliva.* If the use of the remedy is continued, especially in small and repeated doses, combined with opium, so that it is not passed off by the bowels, this mineral induces *salivation*—that is, saliva flows profusely, the gums become tender, red, swollen, and ulcerated on the margins in contact with the teeth. The patient gets rapidly thin during its use. The blood is decomposed, the red corpuscles are rapidly destroyed to the ex-

* The experiments of Dr. George Scott, of Southampton, throw considerable doubts on the hitherto generally received opinion, that calomel in large and purgative doses increases the flow of bile; on the contrary, such doses seem, in the first instance, to diminish the flow of bile; and it is a matter for further experiment to determine whether small and frequent doses of calomel, continued for a length of time, so as to produce the specific action of mercury upon the system, will really ultimately augment the biliary secretion (Beale's *Archives*, vol. i, p. 209).

tent of *one-sixth* or more; the fibrine is diminished by *one-third* of its amount; the albumen by *one-seventh*; and, at the same time, it may become loaded with a fetid matter, the product of decomposition (WRIGHT). "Thus it is an agent of terrible activity, and we may well be cautious how we handle it" (HEADLAND). Its sanative power is believed to consist in controlling or preventing the coagulation of lymph; and for this purpose it is used as an auxiliary to bloodletting. It is only useful in the *sthenic* forms of inflammatory action. It is decidedly hurtful in cases of erysipelas disposed to gangrene, in scrofulous states of the system, in debility, and in cases where the nervous system is in an irritable condition, and the condition of the patient tending to the so-called typhoid state. The specific influence of mercury is recognized by the tenderness of the gums which it induces, by the increase in the quantity of saliva, and by the peculiar mercurial fœtor of the breath. This is the utmost action which should ever be induced. Calomel alone, or calomel combined with opium (a quarter of a grain of the latter with two grains of the former; or a third of a grain of opium with three or four grains of calomel), every three, four, or six hours, is the best form of administration where its influence is rapidly required. But this form of combining mercury with opium should not be persevered in too long, otherwise copious salivation may be induced, which, of all things, ought to be avoided.

Mercury thus employed tends to prevent effusion and favor absorption of effused products. It is advantageously employed in membranous inflammations, and such as go on slowly.

Antimony is antiphlogistic, by tending to increase all the secretions; but particularly those from the skin and lungs. It is especially useful in those *sthenic* inflammations which are rapid, and in which a sudden and powerful action is desired; and also where the direct sanative influence of bloodletting is to be maintained. It is thus indispensable in *croup*, extremely efficacious in *sthenic pneumonia*, and highly useful in *bronchitis*. As an agent to keep up the sanative influence obtained by bloodletting, the action of antimony is invaluable; for when bloodletting has weakened the force of the heart by diminishing the pressure on the vessels, then antimony maintains this diminished pressure in proportion as it weakens the force of the heart. A perseverance in its use produces a watery condition of the blood, diminishing especially the amount of fibrine. The production of nausea is an indication that it has taken sufficient effect.

Alkalies dissolve the fibrine of the blood and retard its formation; and the ingredients of the urine produced out of the destruction of the albuminous compounds of the body, are increased largely in amount by the administration of alkalies. They generally pass out of the body as salts, having combined with acids in the system, and tending to leave behind them an excess of alkali in the blood.

All treatment ought to be judiciously regulated by the knowledge of the tendency of the disease to a spontaneous favorable termination—the accidental symptoms of urgency requiring treatment and control in many cases, rather than the disease itself.

PART II.

METHODICAL NOSOLOGY—SYSTEMATIC MEDICINE, OR THE DISTINCTIONS AND DEFINITIONS, THE NOMEN- CLATURE, AND CLASSIFICATION OF DISEASES.

CHAPTER I.

THE AIM AND OBJECTS OF NOSOLOGY.

NOSOLOGY, regarded as a distinct department of the Science of Medicine, embraces three separate objects of consideration—namely, *First*, the DISTINCTION and DEFINITION of particular diseases, or of the genera and species of diseases; *Secondly*, The NOMENCLATURE of diseases, or the assignment of the names by which they are to be designated, so that each disease may be distinguished and known by an appropriate name; and, *Thirdly*, The ARRANGEMENT or CLASSIFICATION of diseases in some methodic and convenient order, by which they may be distributed into classes, orders, genera, or species. These three divisions of Nosology are respectively known as the DEFINITION, the NOMENCLATURE, and the CLASSIFICATION of diseases. Of these in their order.

I. The Definitions of Diseases.—The first object of Nosology is to obtain such brief enumerations of the peculiar characters of diseases as are sufficient, provisionally, to define them; and the practice of attempting to define diseases so as to lead to their being easily recognized was begun before the time of Galen. In modern times the great advantages that have arisen from establishing definitions in natural history upon fixed and determinate principles, not only of its various objects individually, but also of the groups under which it was found possible to arrange them, suggested to medical men the idea that much advantage might also result to the Science of Medicine from defining diseases, and such groups of diseases as might be found expedient to recognize, under general terms or common names, based upon some fixed and determinate principles. Sydenham recommended that definitions or brief descriptions of diseases should be framed after the model of those that are given of plants; and he lays down various judicious rules for the proper execution of this object in the preface to his work *On Acute Diseases*, first published in 1675. The precepts of Sydenham were never reduced to practice in his day; but about fifty-seven years after his

work *On Acute Diseases* was published, the idea was taken up and acted upon by Franciscus Boissier de Sauvages, a distinguished physician and eminent professor of medicine at Montpellier. He attempted to arrange diseases, as botanists have done plants, into classes, orders, and genera. He endeavored to lay down the characteristic phenomena of each, and to enumerate their principal varieties. The outlines of his nosological system were first published in 1732, and followed thirty years afterwards by his *Nosologia Methodica*—a work which marks an important era in the history of Medicine, as having led to much greater accuracy in the distinction of diseases than was previously observed.

At present the only useful method of defining diseases seems to be an artificial one. It is assumed by nosologists that the proper foundation for the distinction of particular diseases is the occurrence of constant and uniform combinations of morbid phenomena or symptoms, presenting themselves in concurrence or in succession. Thus some of the essentials of a definition are obtained, so that each disease may be marked out by such a brief enumeration of its leading characters as might serve to distinguish it from every other. A series of nosological definitions, more or less correct, may be thus provisionally established, so that the same things are designated by the same terms. Objections have been urged to methods of this kind, on the ground that diseases are unsteady and variable in their character; but the aids to science are now so numerous that physicians are becoming more and more able to distinguish diseases from one another, and to tell by what marks, or upon what grounds, they do so distinguish them. Such are the marks or grounds of distinction by which each disease ought to be defined; and as often as we attempt to establish a distinction among diseases, either the deficiencies or the errors of our definitions will be the more easily perceived; and the attempt will lead to a more accurate consideration of observations previously made, as well as to a greater degree of accuracy in subsequent observations. Definitions of diseases are therefore not only of much service to methodical nosology, but they help to render the diagnosis of diseases more perfect. Pathologists, however, are not agreed as to whether the definitions of diseases should be derived from the external phenomena that present themselves in their course, or from the internal pathological conditions on which these phenomena are supposed to depend; and particularly such of these conditions as consist in lesions or structural alterations discoverable after death.

Cullen was in favor of definitions derived from the symptoms; but he believed that the information derived from pathological anatomy might guide to correct distinctions among diseases. Defining diseases by their supposed proximate causes may lead to error, inasmuch as in many cases these causes are disputable, and may long continue to be so. Whatever principle of defining diseases be adopted, it is absolutely necessary that it should be independent of every theoretical view; for any theory employed, however specious, however much we may be persuaded of its truth, may not appear in the same point of view to others, and may therefore occasion end-

less confusion (CULLEN). If no uniform principle can be laid down for arriving at precise definitions of disease, we must be content with such methods of definition as will serve the main purpose of coupling intelligible general notions regarding the disease with given modes of expression. For example, although we cannot give such a definition of many a disease as will embrace even all the leading phenomena of every case, we may assuredly give such a definition as shall apply with reasonable accuracy to the disease we intend to designate, so that no one may suppose we mean thereby either *small-pox* or the *gout*, when we mean *typhus fever* or *dysentery*; and, in a progressive science like medicine, definitions must always be provisional.

II. The Nomenclature of Diseases.—This, the second object of Nosology, has given rise to many disputes, and has furnished much scope for the display of classical erudition. From the earliest periods of medicine the names imposed upon diseases have been derived from several different sources; but the following considerations have generally regulated the naming of a disease: *First*: Some names have been taken from the part affected—*e.g.*, *peripneumonia*, *podagra*, *ophthalmia*, *dysentery*. *Secondly*: The most characteristic symptoms have furnished the name—*e.g.*, *ileus*, *tenesmus*, *paralysis*, *diarrhœa*, *dyspnœa*, *coma*. *Thirdly*: Some names have been taken from these two circumstances combined—*e.g.*, *cephalgia*, *otalgia*, *cardialgia*, *odontalgia*, *hysteralgia*. *Fourthly*: An alteration of tissue upon which subsequent changes depend being recognized as the essential element of disease, it is named accordingly—*e.g.*, *pleuritis*, *peritonitis*. *Fifthly*: Such alteration not being discovered, the first tangible link in the chain of causation has been used instead—*e.g.*, *melancholia*, *cholera*, *typhus*. *Sixthly*: When a lesion tending to sudden death at once follows the application of the cause, that cause may name the disease—*e.g.*, *lightning*, *prussic acid*, *arsenic*, *burn*, *scald*, *sun-stroke*, *cut*, *stab*, *frost-bite*, &c. *Seventhly*: A considerable number of names of diseases have been derived from some imaginary resemblance to external objects—*e.g.*, *elephantiasis*, *cancer*, *polypus*, *anthrax*, &c. *Lastly*: There are still many names the origin of which it is now easy to trace.

It is obvious, from these statements, that the names of diseases must change as our knowledge changes and becomes more precise; and many diseases which were once named after their symptoms are now called according to the lesion from which most of those symptoms proceed. An apt illustration of this is to be found in *paralysis*, which is no longer regarded as a disease *per se*, but is merely a symptom of several structural alterations of the brain and spinal marrow; and so also *diarrhœa*, which now ought to be almost excluded as a disease from tables of the causes of death.

The progress made in our knowledge of disease from time to time rendered it obvious that some diseases, now only sufficiently recognizable, are different from any other diseases hitherto known. In separating them it became necessary to invent new names for the distinct diseases, or a choice had to be made from amongst those names previously in use. Hence the jumble of Greek, Latin, and

mongrel names which pervades medical nomenclature. The idea also of rendering medical nomenclature *uniform*, by deriving the names of diseases from one source only, or from a certain or mixed combination of sources, has caused many to attempt the reform of medical nomenclature, and especially since morbid anatomy has been so much prosecuted that it might serve as a useful guide in distinguishing the disease or dictating its name.

By some it is maintained that "the name of each disease or species should be so characteristic and significant, that a person slightly acquainted with the language and the subject should, on hearing it, immediately understand what is the nature of the disease it designates" (PLOUCQUET). In this respect the name ought to be composed out of the same elements as the definition of the disease—in fact, it ought to be the definition converted into a name, and derived either from the symptoms of the disease or from the supposed proximate cause. But a name which is expressive only of the nature, seat, or proximate cause of a disease may be erroneous in respect of each of these facts singly, or of all of them together. The history of the nomenclature of fever, especially typhoid, would amply illustrate these statements—*e. g.*, putrid fever, adynamic fever, bilious fever, pythogenic fever, enteric fever, meningo-gastric fever, nervous fever, gastric fever, are mild examples of nomenclature and of confusion, which ought to make a man pause before he attempts to construct a new name. It is inexpedient, also, to abandon (except when unavoidable) the names of distinct diseases received and recognized by our forefathers in the science; or of substituting new ones in their place, without an extreme necessity. Sauvages insists much on this point, and Cullen was of the same opinion. "Words," says the former, "are good only in respect of their signification." In dealing, therefore, with ancient nomenclature, which, for the time being, may appear objectionable, it is surely better to extend, if possible, the signification of the word, name, or term, than to alter it. At the same time it must always be permitted to give new names to new diseases, and to select the best out of those which are in use, when a great number have been used to designate one and the same thing. There are some principles, therefore, which it is well to recognize as influencing the judicious choice of a name. Such names, for example, as involve or attempt to indicate a proximate cause are more liable to lead to error than those which are derived from leading symptoms. If names were to be based on supposed causes, new names of diseases would be required whenever a new hypothesis is started. Look, for example, at the names of typhoid fever, already mentioned, and the systems of Linnæus, Vogel, Pinel, and even Mason Good, will show that medical nomenclature has been repeatedly changed without any urgent necessity; and great inconvenience has especially resulted from incorporating particular and often peculiar pathological doctrines with the language and nomenclature of diseases. So much has this been the case, that the language of medical science has been in danger of becoming "a curious mosaic of the chief speculations of ancient and modern times." The pas-

sion for inventing new terms retards also, in a wonderful degree, the progress of the student of medicine, and tends to involve him in difficulty and doubt.

III. **The Classification of Diseases.**—From time to time physicians have considered it advisable or advantageous to arrange the whole of the diseases they are able to define, and to name, under more or less comprehensive groups. A consideration of the different plans which may be pursued in such arrangements, and of the advantages to be derived from them, forms the third object of Nosology.

It is obvious that any single character, or combination of characters, in respect of which diseases agree with or differ from each other, may be made the basis of methodical arrangement, under a larger or smaller number of divisions, or of higher or lower genera (language of logicians), or of classes, orders, and genera (language of naturalists). By ingenious devices of the mind, the physician or the statist may classify and arrange his knowledge so as to bring it all more readily within his reach for any special purpose,—so as to make it, in fact, more at his disposal—to facilitate and pave the way for further investigation. Such are the legitimate objects and the results of all methodical arrangements. Classification, therefore, being only a method of generalization, there are, of course, several classifications of disease which may be used with advantage for special purposes. The physician, the pathologist, the jurist, the hospital statist, the army medical officer, may each legitimately classify diseases from his own point of view, and for his own purposes, in the way that he thinks the best adapted to facilitate his inquiries, and to yield him general results. The medical practitioner may found his main divisions of diseases on their treatment, as medical or surgical; the pathologist, on the nature of the morbid action or product; the anatomist or the physiologist, on the tissues and organs involved; the medical jurist, on the suddenness, slowness, violent, or unnatural mode of the death; the hospital statist, on the kind of diseases which are treated in its wards; and all of these points of view may give useful and interesting results (FARR).

There is thus no question on which more diversified opinions are legitimately entertained than on that of classification. Although it is the aim of all systematic writers and observers to arrange the objects of study in the most natural order possible, and although diseases are named as if they were individual entities, yet they present so great varieties that they will not admit of that definite and, in many respects, natural species of classification which can be made with objects of natural history. Manifest reasons of convenience and facility for work can therefore be assigned as the great incentive to classification; and numerous reasons exist for classifying diseases in various ways: (1.) Men differ in their estimation of the characters on which different arrangements may be founded. (2.) The facts and phenomena of diseases on which classifications may be made are not all regarded from the same point of view. Most systems are avowedly *artificial*, being arranged with the view to elucidate or support a theory, or otherwise to effect a definite end. For example, by classifying diseases and recording the causes of death,

the most valuable information is obtained relative to the health of the people, or of the unwholesomeness and pestilential agencies which surround them. "We can take this or that disease, and measure not only its destructiveness, but its favorite times of visitation; we can identify its haunts and classify its victims." We are able to trace diseases also as they perceptibly get weaker and weaker, or otherwise change their type, as some have done from time to time. We know from the valuable returns of the Registrar-General, prepared periodically by Dr. Farr, that certain diseases are decreasing, or growing less and less destructive; that certain other diseases have ceased in some measure; while other severe diseases have exhibited a tendency to increase. The advantages, therefore, of adopting some system of classifying diseases, which can be put to such useful practical purposes, must be obvious to every one.

To some extent other systems are *natural* in their arrangement, in so far as they attempt to express or exhibit some of the natural relations which subsist among diseases; but the mere expression of one man's interpretation of peculiarities of disease of the same species, and the elevation of such diseases in a classification as specifically distinct, are apt to be based on insufficient evidence as regards natural relations.

Principles of Classification.—Many systems of Nosology have been adopted from time to time; and as valuable general principles have been adduced from some, the grounds on which diseases have been classified may be briefly described under the following nine divisions, namely:

I. *The nature of the ascertained causes of disease.* On this principle two classes of diseases are recognized, namely,—(1.) Diseases arising from general causes; (2.) Diseases arising from specific causes.

II. *The pathological states or conditions which attend diseases.* The principle of this classification consists in determining alterations of the structure or the chemical composition of parts, from which names are given to the disease—*e. g.*, pleuritis, pneumonia, &c. The distinctions of Sauvages were generally derived from symptomatic and pathological characters, or external symptoms alone; Cullen, following (1792), adopted similar grounds of classification; but with much more comprehensive views than Sauvages, a more lucid order, and a happier simplicity, he excelled in accuracy of definitions all who had gone before him. His descriptions of disease received no coloring from his theories. They are faithful to nature, consistent with the knowledge of his day; and, greatly in advance of his time, his original and inventive mind dwelt much on the causes of disease in all his reasonings and explanations on medical subjects. Aware, however, of the imperfections of the Art of Medicine, he did not attempt to arrange diseases according to their proximate causes, but according to a method founded partly on their symptoms, partly on their causes, and partly on their seats (CURRIE). A methodical arrangement of this kind has generally been considered the most desirable, as being likely to bring together diseases corresponding not only in some very important relations as regards their symptoms, but also in the indications and means of treatment which they sug-

gest and require. But it is obvious that such an arrangement must vary according to the progress of knowledge and of opinion; for a disease which may at present be supposed to depend upon one pathological condition may be found at a future time to proceed from another. Besides, the arrangement involves a principle which tends to separate diseases bearing a striking resemblance to one another in their external phenomena, though depending on different pathological conditions; for example, different species of apoplexy and epilepsy. It is an arrangement, also, which brings together diseases which, though belonging to the same natural family, may be respectively characterized by groups of symptoms that do not bear any very obvious resemblance. Thus the hemorrhages at once bring together apoplexy and hæmoptysis in this classification.

III. *The properties, powers, or functions of an organ or system of organs being deranged*, dictates a classification in which the most prominent effects or phenomena of morbid states are considered as the disease—*e. g.*, palpitation, diarrhœa. It is an arrangement which brings diseases into approximation with one another according to the part of the body principally affected and the function principally disturbed.

When disease consists in perverted powers or functions, it is then denominated a *dynamic* affection or disorder. When it depends on change of structure, it is termed an *organic* lesion or disease.

This third basis of classification is Physiological, and was adopted by Drs. Young and Mason Good in imitation of Ploucquet, of Tübingen. It has been the most popular arrangement of diseases, and perhaps the best adapted for lectures, or for treatises on the practice of physic, because it brings together the different diseases of the same organ, and of those organs most intimately related to one another; but, to profit by the arrangement, the student must be previously instructed in the general doctrines of disease.

IV. The diseases comprehended under the two latter principles of classification are sometimes inaccurately and loosely brought together under the heads of *Structural* and *Functional* diseases. The diseases of function, for instance, being made to embrace the *neuroses*, *hemorrhages*, and *dropsies*; while *inflammation*, *tubercle*, *cancer*, *melanosis*, *hypertrophy*, and *atrophy* are the subordinate classes of the diseases of structure. The diseases of function embrace all those diseases in which the action, the secretion, or the sensation of a part is impaired, without any primary alteration of structure of the organ or tissue affected, so far as our imperfect means of research can ascertain. Thus, *mania*, *cataplexy*, *neuralgia*, are *neuroses* of the brain or other portions of the nervous system. *Colic*, *vomiting*, *diarrhœa*, and *constipation* are *neuroses* of the alimentary canal; and so on of other parts. *Hemorrhage*, or the effusion of blood, and *dropsies*, or an effusion of water into the shut cavities of the body, as that of the head, chest, or abdomen, are also instances of functional disease. Such are the grounds of classification adopted by the late Dr. Williams, of St. Thomas's Hospital, London.

V. A basis of classification has been adopted, founded on the *pathological nature of the different morbid processes*, but the arrange-

ment of the orders and subdivisions are determined by the anatomical arrangement of the textures and organs of the animal body, as originally developed by Bichat.

Such is the principle and mode of classification adopted by Dr. Craigie (1836).

VI. A ground of classification exists, having reference to the *general nature and localization of the morbid states*. It comprehends three classes,—(1.) Diseases which occupy the whole system at the same time, and in which all the functions are simultaneously deranged. These have been named general diseases, such as *fevers*. (2.) Constitutional affections, meaning thereby diseases which display themselves in local lesions in any part, or in several parts of the system, but not in all parts at the same time—*e. g., rheumatism, gout*. (3.) Local morbid processes.

Such is the classification adopted by Dr. Wood, of Philadelphia (1847).

VII. Applying the principles of a *purely humoral pathology*, we have a classification consisting of,—

a. Fevers. b. Dyscrasie—e.g., tabes, chlorosis, scorbutus, dropsy, diabetes, pyæmia, tuberculosis, carcinoma. c. Constitutional diseases, induced by,—(1.) Specific agents; (2.) Vegetable substances. Such is Wunderlich's arrangement of diseases (1852).

VIII. M. de Savignac, Professor of Clinical Medicine at the Naval School of Toulon, propounded (1861) a Nosological arrangement founded on what he believes to be the "elements" of disease. To each of the classes he so defines, the question would at once suggest itself, and require solution, as to what the "element" may be on which the particular class is made to stand alone. He merely sub-joins the word "element" to an adjective formed from the name of each class of diseases. Thus the class Neuroses is distinguished by the *neurotic element*; the class Rheumatalgiæ, by the *rheumatic element*; and so on to the number of fourteen classes. In the formation of orders, genera, or groups of diseases under this classification, no fixed principle can be recognized.

IX. Dr. Stark, of Edinburgh, proposed (1864) an arrangement embracing sixteen classes, namely: (1.) Fevers; (2.) Diseases of the brain, &c.; (3.) Diseases of the heart and organs of circulation; (4.) Diseases of organs of respiration; (5.) Diseases of organs of digestion; (6.) Diseases of urinary organs; (7.) Diseases of organs of generation; (8.) Diseases of organs of locomotion; (9.) Diseases of skin and cellular tissue; (10.) Diseases of uncertain seat; (11.) Malformation; (12.) Debility at birth, and premature birth; (13.) Old age; (14.) Sudden deaths; (15.) Violent or unnatural deaths; (16.) Causes not specified.

None of these nine methods lead to a perfectly philosophical or purely natural classification, because diseases are not yet sufficiently understood to permit us to see clearly their mutual relations; and the best recommendation of any one of them would be a negative one—namely, that of doing the least possible violence to our very imperfect knowledge regarding the natural affinities or alliances of diseases, of which we have at present only a sort of instinctive rec-

ognition. But the tendency of modern investigations by the varied instruments and methods of research tends to prove that many diseases hitherto supposed to be altogether functional are really accompanied with changes of structure, of an anatomical, physical, or chemical kind. It is therefore not unreasonable to anticipate that all the so-called functional maladies will be found to depend upon some concomitant alteration of structure; and when we are unable to detect an alteration either of the solid or fluid parts of the body, in cases where the existence of disease cannot be doubted, we may attribute our failure to the imperfection of our means and instruments of observation, or our modes of using them.

In the present imperfect state of our knowledge, therefore, diseases cannot be philosophically classified, nor arranged according to natural or true pathological relations, dependencies, or alliances. Nevertheless, a great advantage inevitably results from the institution of nosological classification, on account of the necessity which every such attempt imposes on those who engage in it, of marking very accurately the characteristic phenomena of particular diseases; and every one acquainted with the progress of natural history must know that the study of details, and the repeated attempts to systematize them, have mutually promoted and supported each other. It is the same with regard to diseases; and if a Methodical Nosology cannot be rendered perfect, it is a certain proof that, for the time being, the details of which it must be composed are neither accurate nor complete, and are not likely to be so till attempts to observe, investigate, and systematize have made some further progress. Every attempt to reduce to system tends to enlarge our stock of facts; and though we may fail to obtain a perfectly philosophical arrangement, yet the very attempt to attain it must be of advantage, by leading to useful discussions regarding the Pathology and History of diseases (CULLEN). No one could be more convinced than Cullen was, that "perfect division and definition is the summit of human knowledge in every department of science, and requires not only the clearest, but the most comprehensive views, such as (with respect to diseases) we can arrive at only by often repeated attempts and much study." A no less distinguished pathologist—M. Bayle—in discussing the difficulties connected with classification, recommends us "to follow the plan which presents fewest imperfections, remembering that the determination of specific characters is what is most essential in Nosology, arrangement being the least important; for each arrangement will have its defects, will present its deficiencies, and exhibit some forced approximations." Every plan of arrangement ought, therefore, to be accepted for what it is worth, and appreciated at its true value; namely, as to how far it fulfils the object for which it was mainly devised. Cullen, also, in his lectures and in his writings on this subject, everywhere speaks with the utmost modesty and diffidence, and endeavors at all times to impress upon the mind the fact that Nosology, like other branches of medical science, must necessarily be progressive in its advancement; and that it is only by frequent and multiplied trials that it can be brought to any degree of perfection. A perfectly philosophical or natural sys-

tem of classification aims at having the details of its plan to agree in every respect with all the facts as they exist in nature. To effect this end, arrangements, as they *naturally* exist, require to be traced out, not devised. The tracts in which such a pursuit must be followed up, and in which our knowledge is as yet deficient, may be shortly indicated under the following heads, namely: (1.) The affinities or alliances of diseases with each other. (2.) The morbid anatomy of diseased parts. (3.) The communication, propagation, inoculation, generation, development, course, and spontaneous natural termination of diseases. (4.) The connection of the phenomena recognized during life with the facts of morbid anatomy. (5.) The geographical distribution of diseases. (6.) The succession of diseases, so far as they can be traced through past ages; the peculiarities they have exhibited at different periods in the world's history, or within comparatively recent cycles of years. But the time has not yet come for a classification on a basis so comprehensive—simply because the material does not yet exist; and attempts to make so-called *natural* systems of arrangement must end in disappointment, on account of the uncertain and fluctuating data on which they must be based. Such attempts are apt to suggest the serious question, "Whether such Nosology promotes or retards the progress of Medicine?"

Present State and Aim of Nosology.—The most distinguished Physicians and Statists are lending their joint aid to obtain a nomenclature and classification of diseases which can be applied to the wants of the civil and military population in every country. Dr. William Farr devised a system of Nosology which was discussed at several meetings of the Statistical Congress of the Great Powers of Europe, convened for the purpose, amongst other business, of devising and adopting a uniform system of nomenclature for recording diseases and the causes of death from them. The Congress met in Paris on the 10th of September, 1855, when a nomenclature of the causes of death was agreed upon, essentially the same as that used in England and Geneva. At a third Conference, held at Vienna in 1857, a *nomenclature* substantially uniform was agreed upon for adoption in all the states of Europe; and fatal cases were to be registered on a uniform plan. Dr. Farr's system of nomenclature has been in use now for many years by the Registrar-General of this country, and more recently by the Army Medical Department. It was also adopted in previous editions of this text-book; and although it had many imperfections and defects, it was still practically the most useful and authoritative Nosology. "In the English list of names," proposed and adopted by the College of Physicians, "it seemed desirable that as little deviation as possible should be made from those employed by the Registrar-General of England; otherwise his settled plans, and his forms of returns, which have been followed for years, would require to be remodelled; the comparison of future years with past returns would be made difficult and perplexing, if not impossible; and a damaging break would be caused in evidence which becomes more and more

trustworthy and valuable in proportion as it is prolonged and continuous."

The important task of devising a "Provisional Nomenclature and Definition of Diseases," consistent with the progress of medical science, has engaged the attention of a Committee of the Royal College of Physicians of London during the past ten years (1857-1867). After many interruptions and much consideration, the Committee at last completed their work, and submitted a Provisional Nomenclature to the College on the 3d of August, and again on the 28th of November, 1867, when it was unanimously adopted.*

* The circumstances of the origin and progress of this great work are of historical importance, and are thus recorded by the College: "The idea which led to the formation of a general Nomenclature of Diseases originated in a correspondence between Dr. Dumbreck, of the Medical Department of the Army, and Dr. Sibson, respecting the need of such a nomenclature for use in the Army Medical Service. But at the *Comitia majora* of the Royal College of Physicians, held on the 9th July, 1857, it was resolved, on the motion of Dr. Nairne, in consequence of a letter addressed to the College by the Hospitals' Committee of the Epidemiological Society, 'That a Committee be appointed to prepare a Nomenclature of Diseases, and that such Committee have full power to co-operate with other bodies.'

"The following Fellows of the College were appointed members of the Committee by the President of the College: Drs. Mayo, Alderson, Hawkins, Jeaffreson, Pitman, Bence Jones, Risdon Bennet, Munk, Babington, Addison, Nairne, Barker, Budd, Gull, Baly, Barclay, Sibson, Parkes. At the first meeting of the Committee Dr. Sibson was appointed Secretary.

"The following representative members afterwards consented to co-operate in carrying into effect the objects of the Committee: Mr. Stanley, President of the Royal College of Surgeons; Dr. Druitt, Representative of the Master of the Worshipful Society of Apothecaries; Sir John Liddell, Director-General of the Medical Department of the Navy; Dr. Logan, Director-General of the Medical Department of the Army; Sir Ranald Martin, Representative of the East India Company; Dr. Farr, Representative of the Registrar-General; Mr. Simon, Medical Officer of the Privy Council; Mr. Holmes, Secretary of the Hospitals' Committee of the Epidemiological Society.

"The meetings of the Committee were suspended in 1858, in consequence of the passing of the Medical Act of that year, and of the alterations thereby rendered necessary in the constitution and regulations of the College. They were resumed in 1863, and the following members were then or subsequently added to the Committee:

"Sir Thomas Watson, Bart., President of the College of Physicians; Mr. Luke, President of the Royal College of Surgeons; Dr. Bryson, Director-General of the Medical Department of the Navy; Dr. Balfour, Deputy Inspector-General of Hospitals and Head of the Statistical Branch at the Army Medical Board; Dr. Stark, Representative of the Registrar-General of Scotland; Dr. N. M. Burke, Representative of the Registrar-General of Ireland; Dr. Mackay, R.N., Deputy Inspector-General of Fleets; Mr. Moore, Surgeon to the Middlesex Hospitals; and Drs. C. J. B. Williams, Barlow, Arthur Farre, Black, Frederic Weber, Charles West, Chambers, Monro, George Johnson, Quain, Kirkes, Wilks, Bristowe, Henry Thomson, Hermann Weber, Gueneau de Mussy, McWilliam.

"A *Classification* Sub-committee was formed, consisting of—Sir Thomas Watson, Bart. (chairman), Drs. Farr, Barclay, Balfour, C. J. B. Williams, Quain, Sibson, Mr. Simon, and Mr. Holmes.

"A *Definition* Sub-committee was also formed, consisting of—Drs. Barlow, Arthur Farre, West, Chambers, Monro, George Johnson, Barclay, Sibson, Parkes, Kirkes, Wilks, Bristowe, Balfour, Mr. Moore, and Mr. Holmes.

"Mr. Gaskill and Dr. Nairne (Commissioners in Lunacy) attended the meetings of the Committee when the subject of *INSANITY* was under consideration.

"Mr. Cartwright and Mr. Tomes attended the meetings when the diseases of the *TEETH* were under consideration.

"The Latin Nomenclature was prepared by Dr. Henry Thomson, and revised by Dr. Black.

"The French Nomenclature was prepared by Dr. Gueneau de Mussy.

This is a great achievement. The Registrars-General of England, Scotland, and Ireland, the chiefs of the Medical Department of the Army and Navy, and of the British troops in India, have all concurred with others in framing the Nomenclature; and therefore it is not unreasonably expected that greater accuracy, certainty, and uniformity, for comparison, than heretofore will characterize the statistical records of disease, alike in civil life and in the public services. To facilitate the work becoming *international*, the College has translated its nomenclature into Latin, French, German, and Italian equivalents.

The methods of gradually improving the Nomenclature of Diseases in Medicine has some analogy to the methods of gradually improving Representative Reform in Politics. A nomenclature of diseases and a policy of parliamentary representation, judicious and proper a quarter of a century ago, must each eventually give place to the influence of progressive knowledge and power, which invariably come with the rapid movements of the age in which we live. But reform, whether of political representation or medical nomenclature, to be generally acceptable, cannot be the work of one man, nor the accomplishment of a limited period of time. To be on a broad basis and free of prejudice, each work ought to be the combined result of the best men of the time—each man being willing to yield, adapt, and mould his convictions on entering into a mutual arrangement to achieve a common end. Men thus brought together, who differ very seriously as to certain points, may yet, by mutual discussion, come to a definite and reasonable agreement for practical purposes—the terms of the compromise being settled doubtless at the expense of some personal bias, which often has a firmer hold the more imperfect the information of the holder, but which mutual deliberation clears away. Knowing how biased in opinion individual members of professional and political bodies are apt to be, the unanimous adoption of a Provisional Nomenclature by the

"The German Nomenclature was drawn up by Dr. Hermann Weber, and revised by Dr. Frederic Weber.

"The Italian Nomenclature was drawn up by Dr. Frederic Weber.

"The List of Deformities was drawn up by Dr. Arthur Farre.

"The entire work has been edited by the Secretary, Dr. Sibson; with whom Dr. Barclay took part in editing more especially the Medical portion. The Surgical portions of the Nomenclature were prepared and, in conjunction with the Secretary, edited by Mr. Moore and Mr. Holmes.

"Official changes during the period of the existence of the Committee led also to the introduction into it of the following additional members: Mr. Partridge, as President of the Royal College of Surgeons; Drs. Birkett, Owen Rees, Handfield Jones, Basham, Herbert Davies, Guy, Peacock, Wegg, as Censors of the College of Physicians. Dr. Alderson's first official act after his election as President of the College was to appoint Sir Thomas Watson Chairman of the Committee."

These names are the names of men who hold, or have held, the highest place as representative men in the Science of Medicine; and are at once a guarantee of the intellect and practical knowledge which have been brought to bear upon the work.

The preface to the Nomenclature, from the pen of the Chairman of the Committee, is extremely suggestive; and the work ought to be in the hands of every Student of Medicine, and the handbook of every one who has to do with the Registration of Diseases.

London College of Physicians is undoubtedly a great and a bold achievement; and the result is a work beyond all praise.

The plan of the "Provisional Nomenclature" is, first, "*to give an English name to the disease, employing the terms in popular use whenever they are not absolutely inaccurate; and to use only one word, or as few words as possible, in NAMING a disease.*" Definitions have been attached to the English names in some instances only, as where there might be some ambiguity as to the signification which the College desires to attach to them. Thus the definitions have been framed for the purpose of identification only, not as explanations of the phenomena of disease.

The plan is, secondly, *to give a classification based upon anatomical considerations, namely,—GENERAL DISEASES, or such as affect the whole frame, subdivided into Sections A and B;* and LOCAL DISEASES.*

In studying the Science of Medicine systematically, a METHODICAL NOSOLOGY ought to be regarded as a table of reference to aid the student in naming diseases, and so preserving uniformity in his records and diagnosis, and a system to guide him generally in acquiring a knowledge of his profession, especially with reference to the practical questions of the day. The Nosology of the Royal College of Physicians of London ought, therefore, to be accepted simply as a contrivance to aid us in giving the same name to similar conditions of disease, and "for perfecting the statistical registration of diseases, with a view to the discovery of statistical truths concerning their history, nature, and phenomena." Pathology, we know, is yet too young to base a scientific classification upon; and as the Science advances, so must Nosology. But there are many nice questions which always will arise, relative to the nature of diseases, on which it is in vain to expect Physicians and Statists to agree unanimously; and therefore no system even of naming, far less of classifying, the diseases of mankind can we hope to see, otherwise than as a *provisional* one, ready to assimilate itself to the progressive advance of the Science of Medicine. We have every reason to hope that, by the numerous inquisitive researches of the day, Pathology and Nosology will grow even more rapidly than hitherto. The mere enumeration of diseases has almost doubled since Cullen's Nosology was written, while our knowledge of facts relating to disease has greatly more than doubled. Cullen's Nosology became effete and useless at last, under the pressure of increasing knowledge acquired and effected with resources very inferior to

* "Section A comprehends those disorders which appear to involve a morbid condition of the blood, and which present, for the most part, but not all of them, the following characters: They run a definite course, are attended with fever, and frequently with eruptions on the skin, are more or less readily communicable from person to person, and possess the singular and important property of generally protecting those who suffer them from a second attack. They are apt to occur epidemically." They correspond to the *Zymotic diseases* of Dr. Farr's classification.

"Section B comprises for the most part disorders which are apt to invade different parts of the same body simultaneously or in succession. These are sometimes spoken of as constitutional diseases, and they often manifest a tendency to transmission by inheritance." They correspond to the *Constitutional diseases* of Dr. Farr's classification.

those we now possess, and far less extensive. The nomenclature and classification thus adopted by the College is therefore *strictly provisional*; and it would be well if the Colleges of Physicians and Surgeons in Scotland and in Ireland would unite with that of London in this eminently practical work, and appoint committees to communicate with each other in revising and readjusting such nomenclature at the end of every ten years; and so stamp with their united authority the progressive improvements in the Science of Medicine which are capable of being indicated or expressed in the Nomenclature and Definitions of diseases. Such systematic arrangements, if consistent with existing knowledge, never cramp or hamper a man in carrying out scientific investigations; on the contrary, they enable him to see more clearly in what direction his labor must be advanced, and demonstrate more forcibly than otherwise the deficiencies of his knowledge.

The "Provisional Nomenclature" of the Royal College of Physicians of London, comprehended in the following list, has therefore been adopted in the text of this edition (5th); while the synonyms, equivalents, and definitions have also been incorporated, at the places where the diseases are described in the text, throughout both volumes.

CHAPTER II.

TABULAR VIEW OF THE "PROVISIONAL NOMENCLATURE" ADOPTED BY THE ROYAL COLLEGE OF PHYSICIANS OF LONDON.

GENERAL DISEASES.

A.		b. Anginose.
		c. Malignant.*
1. Small-pox.		6. Dengue.
Group A (unmodified).		7. Typhus fever.
Group B (modified).		8. Cerebro-spinal fever. <i>Syn.</i> , Malignant purpuric fever; Epidemic cerebro-spinal meningitis.
Varieties, applicable to both groups:		9. Enteric fever. <i>Syn.</i> , Typhoid fever; and in children is often named Infantile remittent fever.†
a. Confluent.		10. Relapsing fever.
b. Semi-confluent.		11. Simple continued fever.
c. Distinct. <i>Syn.</i> , Discrete.		12. Febricula.
d. Abortive. <i>Syn.</i> , Varicelloid.		13. Yellow fever.
Subordinate Varieties:		14. Plague.
e. Petechial.		14*. Beriberi.
f. Hemorrhagic.		15. Ague. <i>Syn.</i> , Intermittent fever.
g. Corymbose.		Varieties: a. Quotidian.
2. Cow-pox.		b. Tertian.
3. Chicken-pox.		
4. Measles.		
5. Scarlet fever.		
Varieties:		
a. Simple.		

* Scarlet fever occurs occasionally without any rash or sore throat being observed.

† Fevers symptomatic of worms, teething, or other sources of irritation, should not be included under this head.

- Sub-variety:*
Double tertian.
- c. Quartan.*
Sub-variety:
Double quartan.
- d. Irregular.*
- (100^b.) *Brow ague.*
16. Remittent fever.*
17. Simple cholera.
18. Malignant cholera. *Syn.*, Serous cholera; Spasmodic cholera; Asiatic cholera.
 a. Choleraic diarrhœa.
19. Diphtheria.
 a. Diphtheritic paralysis.
20. Whooping-cough.
21. Mumps.
22. Influenza.
23. Glanders.
24. Farcy.
25. Equina mitis. *Syn.*, Grease.
26. Malignant pustule.
27. Phagedœna.
28. Sloughing phagedœna.
29. Hospital gangrene.
30. Erysipelas.
 Varieties:
 a. Simple. Syn., Cutaneous.
 b. Phlegmonous. Syn., Cellulocutaneous.
 c. Diffuse inflammation (of cellular tissue).†
31. Pyæmia.‡
32. Puerperal fever.§
33. Puerperal ephmera. *Syn.*, Weed.

- B.
34. Acute rheumatism. *Syn.*, Rheumatic fever.
 (a.) Subacute rheumatism.
35. Gonorrhœal rheumatism.
36. Synovial rheumatism.
37. Muscular rheumatism.
 Local varieties:
 a. Lumbago.
 b. Stiff neck.
38. Chronic rheumatism.||
39. Acute gout.
40. Chronic gout.
41. Gouty synovitis.||
42. Chronic osteo-arthritis. *Syn.*, Chronic rheumatic arthritis.
43. Purpura.
 Varieties:
 a. Simple.
 b. Hemorrhagic.
44. Scurvy.
 Ergotism.
45. Diabetes. *Syn.*, Diabetes mellitus.
46. Syphilis.
 A. Primary syphilis.
 Varieties:
 Hard chancre.
 Indurated bubo.
 Soft chancre.
 Suppurating bubo.
 Phagedænic sore.
 Sloughing sore.
 B. Secondary syphilis.
 C. Hereditary syphilis.
1. **Local syphilitic affections.

* The malignant local fevers of warm climates are usually of this class.

† In slighter cases, occurring on the surface of the body, this disease is identical with phlegmonous erysipelas.—In registering cases of phlegmonous erysipelas, and of diffuse inflammation arising from injury, surgical operation, or local disease, the cause should be specified.

‡ In returning cases of pyæmia, specify the affected organs.

§ In returning cases of puerperal fever, the more important local lesions, such as peritonitis, effusions into serous and synovial cavities, phlebitis, and diffuse suppuration, should be specified.

|| Cases attended with deposit of lithate of soda are to be returned as chronic gout, and those in which there is marked distortion as chronic osteo-arthritis.

¶ Retrocedent gout is a term applied to cases of gout in which some internal organ becomes affected on the disappearance of the disease from the joints, and should be referred to acute or chronic gout.

** In returning local syphilitic affections, specify whether the case be one of primary syphilis, secondary syphilis, syphilitic deposits, or syphilitic inflammation.—Local syphilitic affections, local cancer, local colloid, and local scrofulous affections, are to be returned in the following order:

Brain.	Supra-renal capsule.	Stomach.	Scrotum.
Spinal cord.	Larynx.	Pylorus.	Testicle.
Nerve.	Bronchi.	Intestines.	Ovary.
Eye.	Lungs.	Rectum.	Fallopian tube.
Eyelid.	Pleura.	Anus.	Uterus.
Orbit.	Mediastinum.	Liver.	Vagina.
Auricle.	Lips.	Hepatic ducts and gall-bladder.	Vulva.
Internal ear.	Mouth.	Pancreas.	Female breast.
Face.	Cheek.	Spleen.	Male mamilla.
Nose.	Jaws.	Peritoneum.	Bone.
Pericardium.	Gum.	Mesenteric glands.	Skull.
Heart.	Tongue.	Kidney.	Joint.
Lymphatics.	Fauces.	Bladder.	Spine.
Lymphatic glands.	Tonsils.	Prostate gland.	Muscle.
Bronchial glands.	Salivary glands.	Penis.	Tendon.
Thyroid gland.	Pharynx.	Prepuce	Cellular tissue.
Thymus gland.	Œsophagus.		Skin.

47. Cancer. *Syn.*, Malignant disease.**Varieties:*

- a. Scirrhus. *Syn.*, Hard cancer.
- b. Medullary cancer. *Syn.*, Soft cancer.
- c. Epithelial cancer. *Syn.*, Carcroid epithelioma.
- d. Melanotic cancer. *Syn.*, Melanosis.
- e. Osteoid cancer.†

1. Local cancer.‡

48. Colloid. *Syn.*, Colloid cancer; Alveolar cancer.

1. Local colloid.§

NON-MALIGNANT TUMORS AND CYSTS.||

Fibrous tumor.

Fibro-cellular tumor.¶

Fibro-nucleated tumor.

Fibro-plastic tumor.**

Myeloid tumor.

Fatty tumor. *Syn.*, Lipoma.

Osseous tumor.

- a. Of bone. *Syn.*, Exostosis.

Varieties: Ivory.

Cancelled.

Diffused.

- b. Of the soft parts.

Cartilaginous tumor. *Syn.*, Enchondroma.

Fibro-cartilaginous tumor.

Glandular tumor. *Syn.*, Adenocèle.

Vascular tumor.

Nævus.

Sebaceous tumor.

Cholesteotoma.

Molluscum.

Warty tumor and warts.

Condyloma.

Cheloid.

Villous tumor.

Simple or barren cysts.

a. Serous.

b. Synovial. *Syn.*, Bursal.

c. Mucous.

d. Suppurating.

e. Sanguineous.

f. Hemorrhagic.

g. Aneurismal.

h. Oily.

i. Colloid or gelatinous.

j. Seminal.

Compound or proliferous cysts.

- a. Complex cystic tumor. *Syn.*, Cysto-sarcoma. With intracystic growths.

b. Cutaneous or piliferous. *Syn.*, Dermoid.

c. Dentigerous.

49. Lupus.

Varieties:

a. Chronic lupus.

b. Lupus exedens.

50. Rodent ulcer.

51. True leprosy. *Syn.*, Elephantiasis Græcorum.

52. Scrofula.

Varieties:

a. Scrofula with tubercle.

b. Scrofula without tubercle.††

1. Local scrofulous affections.

Tubercular meningitis.

Scrofulous ophthalmia.

Scrofulous iritis.

Tubercular pericarditis.

Phthisis pulmonalis.

Scrofulous disease of glands.

Acute miliary tuberculosis.

Tabes mesenterica.

Tubercular peritonitis.††

53. Rickets.

54. Cretinism.

Varieties:

- a. Complete cretinism. *Syn.*, Incurable cretinism.

* In returning cases of cancer in more than one organ, specify in which the disease is primary, and in which secondary.—State also the kind and duration of the disease in each case, and the nature of all operations, with their dates and results.

† Cancer in mucous membranes, when covered by a villous growth, has received the name of Villous cancer.

‡ In returning cases of local cancer, specify the variety of cancer, by adding, after "46," the letter a, b, c, d, or e, according to the nature of the case. They are to be returned in the order specified in the foot-note (**) on preceding page.

§ Cases of local colloid are to be returned in the order specified in the foot-note (**) on preceding page.

|| In order that the malignant and non-malignant growths may appear together, the non-malignant tumors and cysts are inserted here. They should, however, be returned under "Non-malignant tumors" among the local diseases, and they are not, therefore, numbered at this place.

¶ When occurring as a pendulous outgrowth from a mucous surface, it constitutes the chief varieties of Polypus.

** When the fibro-cellular or fibro-plastic tumor, but more especially the latter, slowly involves the adjacent soft structures, and returns after removal, it has received the name of Recurrent fibroid.

†† The constitutional tendency which has received the name of the Scrofulous Diathesis, when unattended by local lesions, is not to be returned under the heading of Scrofula.

‡‡ These and all other cases of local scrofulous affection are to be returned in the order specified in the foot-note (**) on preceding page.

- b. Demi-cretinism.
 c. Incomplete cretinism. *Syn.*, Curable cretinism.
55. *Anæmia.
 56. Chlorosis. *Syn.*, Green-sickness.
 57. *General dropsy.†

DISEASES OF THE NERVOUS SYSTEM.‡

DISEASES OF THE BRAIN AND ITS MEMBRANES.

58. Encephalitis.§
 59. Meningitis.
 1. Inflammation of the dura mater.‖
 2. Inflammation of the pia mater and arachnoid.
 (58¹). 3. *Tubercular meningitis.* *Syn.*, *Acute hydrocephalus.*
 60. Inflammation of the brain.
 61. Red softening (of the brain).
 62. Yellow softening (of the brain).
 63. Abscess (of the brain).
 64. Apoplexy.
 Varieties:
 a. Congestive.
 b. Sanguineous. *Syn.*, Cerebral hemorrhage.
 65. Sunstroke.
 66. Chronic hydrocephalus.
 67. Hypertrophy (of the brain).
 68. Atrophy (of the brain).
 69. White softening (of the brain).
 Syn., Atrophic softening.¶
 (48¹). *Syphilitic disease.*

- (49¹). *Cancer.*
 70. Fibrous tumor.
 71. Osseous tumor.
 (58¹). *Tubercular deposit.*
 a. *Miliary or granular tubercle.***
 b. *Yellow tubercle.*
 72. Parasitic disease.
 73. Diseases of the cerebral arteries.
 1. Degeneration (fatty and calcareous). *Syn.*, Atheroma. Ossification.
 2. Aneurism.
 3. Impaction of coagula.
 1. Thrombosis (local coagulation).
 2. Embolism (coagula conveyed to a distance).

DISEASES OF THE SPINAL CORD AND ITS MEMBRANES.

74. Inflammation.††
 Varieties:
 a. Spinal meningitis.
 b. Myelitis.
 75. Hemorrhage (spinal). *Syn.*, Spinal apoplexy.

* When the cause of this affection has been ascertained, the case should be returned under the head of the Primary disease, the secondary affection being also specified.

† Local dropsies, such as ovarian, and effusions into the serous cavities, as hydrothorax or ascites, when not connected with anasarca, should be returned as local diseases.

‡ ARRANGEMENT OF LOCAL DISEASES.

The Local Diseases have been drawn up in accordance with the following arrangement :

Catarrh.	Passive congestion.	Atrophy.	Cyst.
Inflammation.	Extravasation of blood.	Degeneration.	<i>Scrofula.</i>
Ulcerative inflamm'n.	Hemorrhage.	Fatty and calcareous. <i>Syn.</i> , Atheromatous.	a. <i>With tubercle.</i>
Suppurative "	Dropsy.	Fibroid.	b. <i>Without tubercle.</i>
Plastic "	Fibrinous deposit.	Lardaceous disease.	Parasitic disease.
<i>Pyæmic</i> "	Alteration of dimensions.	<i>Syphilitic disease.</i>	Calculus and concretion.
Rheumatic "	Dilatation.	<i>Cancer.</i>	Malformation.
Gouty "	Contraction.	<i>Colloid.</i>	<i>Injury.</i>
<i>Syphilitic</i> "	Hypertrophy.	Non-malignant tumor.	<i>Foreign body.</i>
<i>Scrofulous</i> "			Functional diseases.
Gonorrhœal "			
Gangrene.			

The diseases printed in italics are to be returned, not among the local diseases, but under the heading referred to by number.

§ This term is to be used only when the precise seat of the inflammation has not been ascertained by post-mortem examination.

‖ This form of inflammation is almost invariably the result of injury or disease of the bones of the skull; in such cases, the injury or disease by which it is caused ought to be specified.

¶ This form of disease is the result of imperfect nutrition, owing to deficient supply of blood, and is in most instances dependent upon mechanical obstruction, or degeneration of the cerebral arteries.

** To be referred to tubercular meningitis.

†† This term is to be used only when the precise seat of the inflammation has not been ascertained by post-mortem examination.

76. Atrophy (spinal). *Syn.*, Tabes dorsalis.
 77. White softening (spinal).
 78. Spina bifida.
 (49¹). *Cancer*.
 79. Non-malignant tumor.

DISEASES OF NERVES.

80. Inflammation.
 81. Atrophy.
 (49.) *Cancer*.
 82. Neuroma.
 83. *Paralysis.
 (107.) 1. *Paralysis of the insane*. *Syn.*,
 General paralysis.
 84. 2. *Hemiplegia.
 85. 3. *Paraplegia.
 86. Locomotor ataxy.
 (79¹). *Progressive muscular atrophy*.
 87. 5. Infantile paralysis.
 6. *Local paralysis. †
 a. Scrivener's palsy.
 7. *Diphtheritic paralysis*.
 8. *Lead palsy*.
 Paralysis from Lathyrus (sativa).

GENERAL DISEASES OF THE NERVOUS SYSTEM.

88. Tetanus.
 89. Hydrophobia.
 90. Infantile convulsions.
 91. Epilepsy.
 Epileptic vertigo. *Syn.*, Petit mal.
 92. *Convulsions.

93. Spasm of muscle.
 94. Laryngismus stridulus. *Syn.*, Spasm of the glottis. Spasmodic croup. Child-crowing.
 95. Shaking palsy.
 96. *Mercurial tremor*.
 97. Chorea. *Syn.*, St. Vitus's dance.
 a. Acute.
 b. Chronic.
 98. Hysteria.
 99. Catalepsy.
 100. Neuralgia.
 Principal varieties:
 a. Facial. *Syn.*, Tic douloureux.
 b. Brow ague. *Syn.*, Hemicrania.
 c. Sciatica.
 101. *Hyperæsthesia.
 102. *Anæsthesia.
 103. *Delirium tremens*.

DISORDERS OF THE INTELLECT, &c.

104. Mania.
 a. Acute mania.
 b. Chronic mania.
 105. Melancholia. ‡
 106. Dementia.
 a. Acute dementia.
 b. Chronic dementia.
 107. Paralysis of the insane. *Syn.*, General paralysis.
 108. Idiocy. (Congenital.)
 109. Imbecility. (Congenital.)
 110. Hypochondriasis.

DISEASES AND INJURIES OF THE EYE.

CONJUNCTIVA.

111. Conjunctivitis. *Syn.*, Ophthalmia.
 112. Catarrhal ophthalmia.
 113. Pustular ophthalmia.
 114. Purulent ophthalmia.
 115. Purulent ophthalmia of infants.
 Syn., Ophthalmia neonatorum.
 (53¹). *Scrofulous ophthalmia*. *Syn.*, *Strumous ophthalmia*.
 116. Exanthematous ophthalmia.
 117. Gonorrhœal ophthalmia.
 118. Chronic ophthalmia.
 119. Edema of the sub-conjunctival tissue. *Syn.*, Chemosis.
 120. Pinguecula.
 121. Pterygium.
 122. Fatty tumors.
 123. Cysticercus telæ cellulose.
 124. Metallic stains.
 a. From nitrate of silver.
 b. From lead.

CORNEA.

125. Keratitis.
 126. Chronic interstitial keratitis.
 127. Keratitis with suppuration. *Syn.*, Onyx.
 128. Ulcer.
 129. Opacity. *Syn.*, Leucoma.
 130. Conical cornea.
 131. Arcus senilis.
 132. Staphyloma.
 133. Entozoa in the anterior chamber.

SCLEROTIC.

134. Scleritis.
 135. Staphyloma.

IRIS.

136. Iritis.
 137. Traumatic iritis.
 138. Rheumatic iritis.
 139. Arthritic iritis.

* When the cause of this affection has been ascertained, the case should be returned under the head of the primary disease, the secondary affection being also specified.

† When the cause of any of these forms of paralysis has been ascertained, it should be stated.

‡ Cases of so-called monomania to be classed under chronic mania or melancholia, according to their character.

- (40¹.) *Syphilitic iritis.*
 (58¹.) *Scrofulous iritis.*
 140. Gonorrhœal iritis.
 141. Sequelæ of iritis.
 142. Congenital defects of iris.

CHOROID AND RETINA.

143. Choroiditis.
 144. Retinitis.
 145. Choroidal apoplexy.
 146. Amaurosis.
 147. Impaired vision.
 148. *Muscæ volitantes.*
 149. Albinism.

VITREOUS BODY.

150. Synchrony.
 151. Various morbid deposits.
 152. Entozoa.

LENS AND ITS CAPSULE.

153. Cataract.
Varieties:
 a. Hard.
 b. Soft.
 c. Fluid.
 154. Congenital cataract.
 155. Traumatic cataract.

GENERAL AFFECTIONS OF THE EYE.

156. Glaucoma.
 157. Hydrophthalmia.
 (49¹.) *Cancer.*
 (58.) *Scrofulous deposit within the eyeball.*
 158. Total disorganization of the eye from injury.

VARIOUS DEFECTS OF SIGHT.

159. Short sight.
 160. Long sight.
 161. Faulty perception of colors. *Syn.,*
 Color blindness.

162. Hemeralopia.
 163. Nyctalopia.
 164. Astigmatism.

DISEASES OF THE LACHRYMAL APPARATUS.

165. Lachrymal obstruction.
 166. Abscess and fistula.
 167. Dacryolith.
 168. Diseases of the lachrymal gland and its ducts.

DISEASES OF THE EYELIDS.

169. Inflammation.
 170. Hordeolum.
 171. Abscess in the Meibomian glands.
 172. Epicanthis
 173. Entropium.
 174. Ectropium.
 175. Trichiasis.
 176. Madarosis. *Syn.,* Loss of the eyelashes.
 177. Tarsal ophthalmia.
 178. Blepharospasmus.
 (49.) *Cancer.*
 179. Cyst of the lids.
Phthiriasis.

DISEASES OF THE PARTS WITHIN THE ORBITS.

180. Abscess in the orbit.
 181. Strabismus.
 182. Protrusion of the eyeball. *Syn.,*
Proptosis.
 (277.) *Ex-ophthalmic bronchocele.*
 (248.) *Orbital aneurism.*
 (49.) *Cancer.*
 183. Non-malignant tumor.
 184. Hydatid tumor in the orbit.
 185. Affections of orbital nerves.
(Injuries of the eye are given at p.
 197.)*

DISEASES OF THE EAR.

AURICLE.

186. Gouty and other deposits.
 187. Hæmatoma auris.
 (49¹.) *Cancers.*
 188. Non-malignant tumor.
 (821, &c.) *Cutaneous affections.*
 189. Malformations.
 (1007.) *Injuries.*

EXTERNAL MEATUS.

190. Inflammation.
 a. Acute.
 b. Chronic.
 191. Abscess.
 192. Accumulation of wax.
 193. Polypus.
 194. Sebaceous tumor. *Syn.,* Molluscous.

195. Exostosis.
 (1007.) *Foreign bodies.*

MEMBRANA TYMPANI.

196. Inflammation.
 197. Ulceration.
 198. Perforation.
 (1007.) *Injuries.*

EUSTACHIAN TUBE.

199. Obstruction.

TYMPANUM.

200. Disease of the mucous membrane.
 201. " " ossicles.
 202. " " mastoid cells.

* When any of these affections implicate the brain, carotid artery, or lateral sinus, the fact should be stated.

INTERNAL EAR.

203. Organic disease.
204. Necrosis of petrous bone.
205. Deafness.

- a. Functional or nervous.
b. From disease.
c. Deaf-dumbness.
(49¹.) *Cancer*.*

DISEASES OF THE NOSE.

206. Hypertrophy. *Syn.*, Lipoma.
207. Wart.
208. Sebaceous cyst.
(49¹.) *Cancer of the skin*.
(51¹.) *Lupus*.
209. Ozena.
210. Abscess of the septum.
210^a. Ulceration of the pituitary membrane.
211. Perforation of the septum.
212. Epistaxis.
213. Hypertrophy of the pituitary membrane.

214. Polypus nasi.
Varieties:
a. Gelatinous.
b. Fibrous.
Naso-pharyngeal polypus.
215. Non-malignant tumors of the septum.
216. Rhinoliths.
(1008.) *Foreign bodies*.
(49¹.) *Cancer*. *Syn.*, *Malignant polypus*.
Loss or perversion of sense of smell.

DISEASES OF THE CIRCULATORY SYSTEM.

DISEASES OF THE HEART AND ITS MEMBRANES.

DISEASES OF THE PERICARDIUM.

- 216^a. Pericarditis.
217. Suppurative pericarditis.
(53¹.) *Tubercular pericarditis*.
218. Adherent pericardium.†
219. Dropsy.
(49¹.) *Cancer*.
220. Malformations.

DISEASES OF THE ENDOCARDIUM.

221. Endocarditis.‡
222. Valve-disease.
1. Aortic.
2. Mitral.
3. Of pulmonary artery.
4. Tricuspid.
Varieties:
a. Vegetations.
b. Fibroid thickening.
c. Atheromatous and calcareous degeneration.
d. Aneurism.
e. Laceration.
f. Simple dilatation of orifice.
g. Malformations.
Obstruction to the circulation, or Regurgitations should be specially noted when they accompany the valve-disease.
223. Fibrinous concretions in the cavities of the heart.§

DISEASES OF THE MUSCULAR STRUCTURE OF THE HEART.

223. Myocarditis.
224. Abscess.||
225. Hypertrophy.
a. Of left side.
b. Of right side.
226. Dilatation.
a. Of left side.
b. Of right side.
227. Atrophy.
228. Excess of fat.
229. Fatty degeneration.
230. Fibroid degeneration.
231. Aneurism.
232. Acute aneurism.
233. Rupture.¶
(49¹.) *Cancer*.
234. Entozoa.
235. Disease of the coronary arteries.
236. Malformations.**
237. Cyanosis.
238. *Injuries of the heart*.
239. ††Angina pectoris.
240. ††Syncope.
241. ††Palpitation and irregularity of the heart.

DISEASES OF THE BLOODVESSELS.††

DISEASES OF THE ARTERIES.

242. Arteritis.
243. Degeneration. Fatty and calcareous.
Syn., Atheroma. Ossification.

* When any of these affections implicate the brain, carotid artery, or lateral sinus, the fact should be stated.

† Including under this term partial adhesions and calcareous and ossific deposits.

‡ In returning such cases state, if possible, the valve or valves affected.

§ Cases are to be returned under this head only when the condition has evidently existed during life, and is believed to have been the cause of death.

¶ Abscess dependent on pyæmia should be referred to that disease.

‡ In returning cases of aneurism and rupture, the situation ought to be stated.

** State which, according to list, page 201.

†† When the cause of this affection has been ascertained, the case should be returned under the head of the primary disease, the secondary affection being also specified.

‡‡ The vessel affected should in all cases be specified.

244. Narrowing and obliteration.
 246. Occlusion.
 a. From compression.
 b. From impaction.
 1. Thrombosis (local coagulation).
 2. Embolism (coagula conveyed from a distance).
 247. Dilatation.
 248. Aneurism.
 In returning such cases, state whether the aneurism be—
 a. Fusiform.
 b. Saccular, or
 c. Diffused (sac formed by the surrounding tissue).
 249. Rupture of artery.
 a. From disease of artery.
 b. From disease external to artery.
 250. Partial rupture of artery. *Syn.*, Dissecting aneurism.
 251. Traumatic.
 252. Arterio-venous aneurism.
 253. Aneurismal varix.
 Varieties:
 a. Traumatic.
 b. Spontaneous.
 254. Varicose aneurism.
 Varieties:
 a. Traumatic.
 b. Spontaneous.
 255. Cirroid aneurism. *Syn.*, Arterial varix.

256. Aneurism by anastomosis.
 257. Malformation.
 a. Constriction or occlusion of the commencement of the descending aorta (originating in partial malformation).
 (1049^a.) *Injuries of arteries.*
 (1049^{a1}.) *Contusion.*
 (1049^{a2}.) *Laceration. Syn., Partial rupture.*
 a. Of outer coat.
 b. Of inner coat.
Wound.

DISEASES OF VEINS.

- 257^a. Phlebitis.
 Varieties:
 a. Adhesive.
 Phlegmasia dolens.
 b. Suppurative.
 258. Phlegmasia dolens.
 258. Fibrinous concretions in the veins.
 259. Obstruction.
 260. Obliteration.
 261. Phlebolithes.
 262. Varicose veins.
 263. Nævus vascularis.
 (1049^b.) *Injuries of veins.*
 (1049^{b1}.) *Rupture, without external wound.*
 (1049^{b2}.) *Wound of vein with entrance of air.*

DISEASES OF THE ABSORBENT SYSTEM.

264. Inflammation of lymphatics.
 265. Suppuration of lymphatics.
 266. Inflammation of glands.
 267. Suppuration of glands.
 268. Hypertrophy of glands.
 a. Chronic enlargement of glands.
 269. Atrophy of glands.
 270. Lymphatic fistula.
 (1182.) *Foreign bodies and concretions.*
 271. Obstruction of the thoracic duct.*
 272. Obstruction, obliteration, and varicosity of lymphatics.
 278. Bursting of lymphatics.
 (49.) *Syphilitic bubo.*
 (49.) *Syphilitic inflammation of glands.*
 (50.) *Cancer.*
 (53.) *Scrofulous disease of glands.*
 Suppuration.
 (1183.) *Wounds of lymphatics.*

Diseases of the Bronchial Glands.

- (338.) *Inflammation.*
 (339.) *Abscess.*
 (340.) *Enlargement.*
 (49¹.) *Cancer.*
 (341.) *Non-malignant growth.*
 (53¹.) *Tubercle.*

DISEASES OF DUCTLESS GLANDS.

DISEASES OF THE THYROID GLAND

274. Inflammation.
 a. Acute.
 b. Chronic.
 275. Goitre.
 276. Cyst.
 277. Exophthalmic bronchocele.
 278. Pulsating bronchocele.
 (49¹.) *Cancer.*

DISEASES OF THE THYMUS GLAND.

279. Hypertrophy.

- (50.) *Cancer.*
 280. Non-malignant tumor.

DISEASES OF THE SUPRA-RENAL CAPSULES.

281. Suppuration.
 (49¹.) *Cancer.*
 (53.) *Tubercular degeneration.*
 282. Addison's disease. *Syn.*, Bronze skin. Melasma Addisoni.

* The cause of the obstruction should be stated.

DISEASES OF THE RESPIRATORY SYSTEM.

DISEASES OF THE RESPIRATORY SYSTEM NOT STRICTLY LOCAL.

283. Hay asthma.
 (22.) *Influenza*.
 (20.) *Whooping-cough*.
 284. Croup.
 (19.) *Diphtheria*.
 (989.) **Asphyxia*.

DISEASES OF THE NOSTRILS.

285. Coryza. *Syn.*, Nasal catarrh.

DISEASES OF THE LARYNX.

290. Inflammation of the epiglottis.
 291. Ulceration of the epiglottis.
 292. Laryngeal catarrh.
 293. Laryngitis.
 a. Acute.
 b. Chronic.
 294. Ulcer.†
 295. Abscess.
 296. Edema of the glottis.
 297. Necrosis of cartilage (see the previous Note).
 298. Contraction.
 (49¹.) *Epithelial cancer*.
 299. Warty growth.
 300. Polypus.
 301. Cyst.
 (1086.) *Foreign bodies in the larynx*.

FUNCTIONAL AFFECTIONS OF THE LARYNX.

302. *Aphonia.
 303. *Paralysis of the glottis.
 304. *Spasm of the glottis.
 (94.) *Laryngismus stridulus*.

DISEASES OF THE TRACHEA AND BRONCHI.

305. Bronchial catarrh.
 306. Bronchitis.
 a. Acute.
 b. Chronic.
 307. Ulcer.
 308. *Casts of the bronchial tubes.
 309. Necrosis of the cartilages of the trachea.‡
 310. Dilatation.
 311. Contraction.
 (49¹.) *Cancer*.

312. Non-malignant Tumor.
 (53¹.) *Tubercle*
 (1086.) *Foreign body in the bronchi*.
 318. Asthma.

DISEASES OF THE LUNG.

314. Pneumonia.
 Variety:
 a. Lobular.‡
 315. Abscess.
 (28.) *Pyæmic inflammation and abscess*.
 316. Gangrene.
 317. *Passive congestion.
 318. *Pulmonary extravasation.
 Pulmonary apoplexy.
 319. *Edema.
 320. Cirrhosis.
 321. Emphysema.
 a. Vesicular.
 b. Interlobular.
 322. Atelectasia.
 323. *Collapse.
 (48¹.) *Syphilitic deposit*.
 (49¹.) *Cancer*.
 (53¹.) *Phthisis*.
 (53¹.) *Acute miliary tuberculosis*.
 324. Acute pneumonic phthisis.
 325. Hydatid.
 325^a. Chronic pneumonic phthisis.
 (1042-46.) *Injuries*.
 (1086.) *Foreign bodies*.
 326. Millstone makers' phthisis.
 327. Grinders' asthma.
 328. Miners' asthma.

DISEASES OF THE PLEURA.

329. Pleurisy.
 330. Chronic pleurisy.
 331. Empyema.
 332. Adhesions, including thickening and ossification.
 333. *Hydrothorax.
 334. Pneumothorax.
 (49¹.) *Cancer*.
 335. Non-malignant tumor.
 (53¹.) *Tubercular pleurisy*.
 (1045.) *Injuries*.

DISEASES OF THE MEDIASTINUM.

336. Abscess.
 (49¹.) *Cancer*.
 337. Non-malignant tumor.
 (279.) *Diseases of the thymus gland*.

* When the cause of this affection has been ascertained, the case should be returned under the head of the primary disease, the secondary affection being also specified.

† When chronic laryngitis, ulcer of the larynx, or necrosis of the cartilage (*see below*), is due to phthisis or syphilis, the terms *Syphilitic* or *Phthisical* should be prefixed to the designation of the disease, and the case ought to be returned under the head of the primary affection.

‡ When this affection is due to phthisis or syphilis, the terms *Syphilitic* or *Phthisical* should be prefixed to the designation of the disease, and the case ought to be returned under the head of the primary affection.

§ The term *Secondary* has been applied to pneumonia when it occurs as a complication of some other disease; such cases ought to be returned under the head of the primary affection.

DISEASES OF THE BRONCHIAL GLANDS.

338. Inflammation.
339. Abscess.
340. Enlargement.

- (49¹.) *Cancer*.
341. Non-malignant tumor.
(58¹.) *Tubercle*.

DISEASES OF THE DIGESTIVE SYSTEM.

DISEASES OF THE LIPS.

The affected lip ought to be specified.

342. Ulcer.
(48¹.) *Syphilitic ulcer*.
343. Fissures.
(49¹.) *Cancer*.
(58¹.) *Strumous hypertrophy*.
344. Cyst.
345. Malformations.
346. Hare-lip.

DISEASES OF THE MOUTH.

347. Stomatitis.
348. Ulcerative stomatitis.
349. Thrush. *Syn.*, Aphtha. Vesicular stomatitis.
350. Parasitic thrush. *Syn.*, Parasitic aphthæ.
351. Abscess of the cheek.
352. Cancrum oris. *Syn.*, Gangrenous stomatitis.
353. Cysts of the cheek.
354. Ranula.
(49¹.) *Cancer*.

DISEASES OF THE JAWS (exclusive of the Alveoli).

355. Adhesion of the jaws by cicatrix.
356. Abscess of the antrum.
(49¹.) *Cancer*.
357. Fibrous tumor.
358. Myeloid tumor.
359. Osseous tumor.
Hypertrophy of the bones of the face.
360. Cartilaginous tumor.
361. Vascular tumor.
362. Cyst.

DISEASES, MALFORMATIONS, AND INJURIES OF THE TEETH, GUMS, AND ALVEOLI.

363. Teething.*

DISEASES OF THE DENTAL TISSUE.

364. Caries.
365. Necrosis.
366. Exostosis.
367. Absorption.

DISEASES OF THE DENTAL PULP.

368. Irritation.

369. Inflammation.
370. Ulceration.
371. Gangrene.
372. Granulation or polypus.
373. Calcification.

DISEASES OF THE DENTAL PERIOSTEUM.

374. Inflammation.
375. Gum-boil.
376. Chronic thickening.
377. Rheumatic inflammation.

DISEASES OF THE GUMS.

378. Inflammation.
379. Ulceration.
380. Hypertrophy.
381. Atrophy.
382. Induration (in infancy).
(50¹.) *Cancer*.
383. Non-malignant tumor.
a. Polypus.
b. Cartilaginous tumor.
c. Vascular tumor.
384. Epulis.

DISEASES OF THE ALVEOLI.

385. Inflammation.
386. Necrosis.
387. Caries.
388. Exostosis.
389. Dentigerous cyst.
390. Absorption.

SPECIFIC DISEASES AFFECTING THE DENTAL PERIOSTEUM, GUMS, OR ALVEOLI.

391. Mercurial inflammation.
392. Phosphoric inflammation and necrosis.
(902^c.) *Blue gum from lead*.
(45.) *Scurvy*.

IRREGULAR DENTITION.

- Irregularity in the time of eruption of the—
393. Temporary teeth.
394. Permanent teeth.
Irregularity in the position of the—
395. Temporary teeth.
396. Permanent teeth.
Irregularity of the number of the—
397. Temporary teeth.
398. Permanent teeth.
Irregularity in the form of the—

* Any affection, such as convulsions and paralysis, induced by this condition, should be specified.

- 399. Temporary teeth.
- 400. Permanent teeth.
- Abnormal development of the—
- 401. Dental tissue.
- 402. Enamel.
- 403. Dentine.
- 404. Cementum.
- 405. Alveolar portions of the jaws, in size.
- 406. Alveolar portions of the jaws, in form.
- 407. Defective growth of lower jaw.
- 408. Mechanical injuries of the alveoli and dental periosteum.
 - a. Hemorrhage.
 - b. Fracture.
- 409. Mechanical injuries of the teeth.
 - a. Fracture.
 - b. Dilaceration.
 - c. Dislocation.
 - d. Friction.

DISEASES OF THE TONGUE.

- 410. Glossitis.
- 411. Ulceration.
- 412. Aphthous ulcer.
- 413. Abscess.
- 414. Hypertrophy.
 - (48¹.) *Primary syphilis.*
 - (48¹.) *Secondary syphilis.*
 - (49¹.) *Epithelial cancer.*
- 415. Vascular tumor.
- 416. Tongue-tie.
 - (87.) **Paralysis.*

DISEASES OF THE FAUCES AND PALATE.

- 417. Sore throat.
- 418. Relaxed throat.
- 419. Ulcerated throat.
- 420. Quinsy. *Syn.*, Cynanche tonsillaris.
- 421. Tonsillitis.
- 422. Sloughing sore throat. *Syn.*, Putrid sore throat. Cynanche maligna.†
 - (19.) *Diphtheria.*
- 423. Enlarged tonsils.
 - (49¹.) *Cancer of tonsils.*
 - (58¹.) *Scrofulous diseases of tonsils.*
- 424. Elongated uvula.
- 425. Perforation of the palate.
- 426. Stricture of the fauces.
 - (48¹.) *Syphilitic affection of fauces and tonsils.*
 - (49¹.) *Cancer.*
- 427. Non-malignant tumor.
 - a. Fibro-cellular tumor.
 - b. Fibro-cystic tumor.
- 428. Cleft palate.

DISEASES OF THE PHARYNX.

- 429. Pharyngitis.
- 430. Ulcer.
 - a. Superficial ulcer.
 - b. Perforating ulcer.
- 431. Abscess.
- 432. Sloughing.
- 433. Adhesion of soft palate.
- 434. *Dilatation.
 - (48¹.) *Syphilitic affection.*
 - (49¹.) *Cancer.*
 - (1087.) *Foreign bodies.*
 - (87.) **Paralysis.*

DISEASES OF THE SALIVARY GLANDS.

- 435. Inflammation.
- 436. *Salivation. *Syn.*, Ptyalism.
- 437. Abscess.
- 438. Salivary fistula.
 - (21.) *Mumps.*
 - (49¹.) *Cancer.*
- 439. Non-malignant tumor.
- 440. Salivary calculus.‡

DISEASES OF THE ESOPHAGUS.

- 441. Esophagitis.
- 442. Ulceration.
- 443. *Perforation.
- 444. *Stricture.
 - (49¹.) *Cancer.*
 - (1088.) *Foreign bodies.*
- 445. Malformations.
 - (88.) **Paralysis.*
- 446. Dysphagia.

DISEASES OF THE STOMACH.

- 447. Gastritis.
 - (916, &c.) a. *From irritant poisons.*
- 448. Chronic ulcer.
- 449. Hæmatemesia.
- 450. Perforation.§
- 451. *Dilatation.
- 452. *Stricture.
- 453. Gastric fistula.
- 454. Hernia.
 - (49¹.) *Cancer.*
 - (50¹.) *Colloid.*
- 455. Non-malignant tumor.
- 456. Sarcinæ.
 - (1061–63.) *Injuries to the stomach.*
 - (1065.) *Foreign bodies.*
- 457. Laceration (spontaneous).
- 458. Dyspepsia.
- 459. Gastrodynia.
- 460. Pyrosis.
- 461. *Vomiting.

* When the cause of this affection has been ascertained, the case should be returned under the head of the primary disease, the secondary affection being also specified.

† This affection must be distinguished from malignant scarlet fever.

‡ Whenever any of the affections of the mouth, throat, or parts connected therewith depend on syphilis, scurvy, local irritants, or any other specific cause, the fact should be stated.

§ The cause of the perforation, when ascertained, should be stated.

DISEASES OF THE INTESTINES.

- 462. Enteritis.
- 463. Typhlitis.
- 464. Dysentery.
- 465. Ulceration.
- 466. Perforation.
- 467. Abscess in the sub-peritoneal tissue.
- 468. Fecal abscess.
- 469. Fistula.

- a. Fecal fistula. *Syn.*, Artificial anus.

- (557.) Vesico-intestinal fistula.

- 470. Hemorrhage.

- 471. Melæna.

- 472. *Dilatation.

- 473. *Tympanites.

- 474. *Obstruction.

- 475. Stricture.

- 476. Intussusception.

- 477. Internal strangulation.

- a. Mesenteric.

- b. Mesocolic.

- 478. Hernia.

- a. Reducible.

- b. Irreducible.

- c. Obstructed.

- d. Inflamed.

- e. Strangulated.

- 1. Diaphragmatic.

- 2. Epigastric.

- 3. Ventral.

- 4. Umbilical.

- 5. Lumbar.

- 6. Inguinal.

- a. Oblique.

- b. Direct.

- c. Incomplete.

- d. Scrotal.

- e. Congenital.

- 7. Femoral.

- 8. Obturator.

- 9. Perineal.

- 10. Pudendal.

- 11. Vaginal.

- 12. Ischiatic.

- 479. Diseases of hernial sacs.

- a. Inflammation.

- b. Fibrinous effusion with closure.

- c. Suppuration.

- d. Dropsy.

- e. Movable bodies.

- f. Laceration.

- (491.) Cancer.

- (501.) Colloid.

- 480. Non-malignant tumor.

- a. Polypus.

- 481. Worms.

- (1066.) Concretions.

- (1066.) Foreign bodies.

- (1061-63.) Injuries.

- 482. Diarrhœa.

- (17.) Simple cholera.

- (18.) Malignant cholera.

- a. Choleraic diarrhœa.

- 483. *Paralysis.

- 484. Colic.

- (902a.) Lead colic.

- 485. Constipation.

DISEASES OF THE RECTUM AND ANUS.

- 486. Ulcer.

- 487. Abscess.

- 488. Fistula in ano.

- (558.) Recto-vesical fistula.

- (586.) Recto-urethral fistula.

- (670.) Recto-vaginal fistula.

- 489. Hæmorrhoids.

- a. Internal.

- b. External.

- 490. Hemorrhage from rectum.

- 491. Fissure of the anus.

- 492. Prolapsus.

- 493. *Stricture.

- (49.) Cancer of the rectum.

- (49.) Cancer of the anus.

- Syphilis of rectum.

- Condyloma of anus.

- 494. Non-malignant tumor of the rectum.

- a. Polypus.

- (1072, 78.) Injuries.

- (1079.) Foreign bodies.

- 495. Neuralgia.

- 496. Spasm of the sphincter ani.

- 497. Pruritus ani.

DISEASES OF THE LIVER.

- 498. Hepatitis.

- 499. Abscess.†

- (28.) Pyæmic inflammation and abscess.

- 500. Acute atrophy.

- 501. Simple enlargement. *Syn.*, Congestion of the liver.

- 502. Thickening of the capsule.

- 503. Cirrhosis.

- 504. Fatty liver.

- 506. Fibroid deposit.

- 505. Lardaceous liver. *Syn.*, Amyloid disease of the liver. Waxy liver.‡

- (48.) Syphilitic deposit.

- (49.) Cancer.

- (50.) Colloid.

- 507. Non-malignant tumor.

- 508. Cyst.

- (531.) Tubercle.

- 509. Hydatid.

- (1061-63.) Injuries.

- 510. Jaundice. *Syn.*, Icterus.

- 511. Obstruction of vena portæ.

* When the cause of this affection has been ascertained, the case should be returned under the head of the primary disease, the secondary affection being also specified.

† When abscess of the liver is associated with dysentery, injury, or any other condition, the fact should be stated.

‡ Such cases have been described under the name of Scrofulous disease of the liver.

DISEASES OF THE HEPATIC DUCTS AND GALL-BLADDER.

- 512. Inflammation.
- 513. Ulcer.
- 514. Perforation.
 - a. Biliary fistula.
- 515. Obstruction.
 - (49¹.) *Cancer*.
- 516. Gallstones.
 - a. Passage of gallstones through duct.
- (1061-63.) *Injuries*.

DISEASES OF THE PANCREAS.

- 517. Abscess.
- 518. Obstruction of the duct.
 - (49¹.) *Cancer*.
 - (50¹.) *Colloid*.
- 519. Calculi.

DISEASES OF THE SPLEEN.

- 520. Splenitis.
- 521. Abscess.
 - (28.) *Pyæmic inflammation and abscess*.
- 522. Congestion. *Syn.*, Ague cake.
- 523. Fibrinous deposit.
- 524. Hypertrophy.
 - a. Leucocythæmia.

DISEASES OF THE URINARY SYSTEM.**DISEASES OF THE KIDNEY.**

- 534. Bright's disease. *Syn.*, Albuminuria.
 - 1. Acute Bright's disease. *Syn.*, Acute albuminuria. Acute desquamative nephritis. Acute renal dropsy.
 - 2. Chronic Bright's disease. *Syn.*, Chronic albuminuria.
 - Sub-divisions:*
 - a. Granular kidney. *Syn.*, Contracted granular kidney. Chronic desquamative nephritis. Gouty kidney.
 - b. Fatty kidney.
 - c. Lardaceous kidney. *Syn.*, Amyloid disease. Waxy disease.
- 535. Suppurative nephritis.
- 536. Abscess.
- 537. Pyelitis.
- 538. Fibrinous deposit.
- 539. Hydronephrosis.
- 540. Hypertrophy.
- 541. Atrophy.
 - (49¹.) *Cancer*.
- 542. Non-malignant tumor.
- 543. Simple cyst.
- 544. Urinary cyst (from injury).
 - (53¹.) *Tubercle*.
- 545. Entozoa

- 525. Lardaceous spleen. *Syn.*, Amyloid disease. Waxy spleen.
 - (49¹.) *Cancer*.
 - (50¹.) *Colloid*.
 - (53¹.) *Tubercle*.
- 526. Hydatid.
 - (1058.) *Rupture*.

DISEASES OF THE PERITONEUM.

- 527. Peritonitis.
 - (713.) a. *Puerperal peritonitis*.
 - b. Chronic peritonitis.
 - c. Suppurative peritonitis.
 - (53¹.) d. *Tubercular peritonitis*.
 - e. Adhesions of peritoneum.
- 528. *Ascites.
 - (49¹.) *Cancer*.
 - (50¹.) *Colloid*.
- 529. Hydatid.
 - (1059-62.) *Injuries*.

DISEASES OF THE MESENTERIC GLANDS.

- 530. Inflammation.
- 531. Abscess.
- 532. Enlargement.
 - (49¹.) *Cancer*.
- 533. Non-malignant growth.
 - (53¹.) *Tubercle*.
 - (58¹.) *Tubes mesenterica*.

a. Hydatid.**b. Eustrongylus gigas.**

- 546. Calculus.
- 547. Calculus in the ureter.
- 548. Malformations.
 - (1061-63.) *Injuries*.
- 549. *Hæmaturia renalis.
- 550. *Suppression of urine. *Syn.*, Ischuria renalis.
 - (47.) *Diabetes*. *Syn.*, Diabetes mellitus.
- 551. *Diuresis.
- 552. Movable kidney.

DISEASES OF THE BLADDER.

- 553. Cystitis. *Syn.*, Catarrh of the bladder.
 - a. Acute.
 - *b. Chronic.
- 554. Ulceration.
- 555. Suppuration.
- 556. Sloughing.
- 557. Vesico-intestinal fistula.
- 558. Recto-vesical fistula.
- 559. Vesico-vaginal fistula.
- 559. Hypertrophy.
- 560. *Distension.
 - a. Sacculated bladder.
 - b. Rupture.
- 561. Inversion.
- 562. Extroversion.

* When the cause of this affection has been ascertained, the case should be returned under the head of the primary disease, the secondary affection being also specified.

563. Hernia.
(49.) *Cancer*.
564. Fibrous tumor.
565. Villous tumor.
566. Calculus.
 a. Uric acid.
 b. Urate of ammonia.
 c. Uric oxide. *Syn.*, Xanthic oxide.
 d. Oxalate of lime.
 e. Cystic oxide.
 f. Phosphate of lime.
 g. Triple phosphate.
 h. Fusible.
 i. Carbonate of lime.
 k. Fibrinous.
 l. Urostealith.
 m. Blood calculus.
 Foreign bodies.
567. *Hæmaturia (vesical).
568. *Paralysis.
569. *Irritability.
570. *Spasm.
571. Neuralgia.
572. *Incontinence of urine.
573. *Retention of urine.

DISEASES OF THE PROSTATE GLAND.

574. Inflammation.
 a. Acute.
 b. Chronic.
575. Ulceration.
576. Abscess.
577. Atrophy.
(49.) *Cancer*.
578. Non-malignant tumor. *Syn.*, Enlarged lobe of prostate.
579. Cyst.
(71.) *Tubercle*.
580. Calculi.

GONORRHEA AND ITS COMPLICATIONS.

581. Gonorrhœa.
 a. In male.
 b. In female.
582. Balanitis.
 Herpes preputialis.
583. Phimosiis.
584. Paraphimosiis.
585. Condyloma.
 a. In male.
 b. In female.
586. Gleet.
587. Lacunar abscess.
588. Bubo.
 Prostatic abscess.
589. Epididymitis. *Syn.*, Gonorrhœal orchitis.
 a. Abscess.
590. Abscess of the spermatic cord.
(828.) *Inflammation of ovary*.
591. Abscess of the vulva.
(117.) *Gonorrhœal ophthalmia*.
(140.) *Gonorrhœal iritis*.
(86.) *Gonorrhœal rheumatism*.

DISEASES OF THE MALE URETHRA.

592. Stricture.†
 a. Organic.
 b. Traumatic.
 c. Spasmodic.
 d. Inflammatory.
593. Ulcer.
594. Urinary abscess.
595. Urinary fistula.
596. Recto-urethral fistula.
597. Extravasation of urine.
598. Impacted calculus.
 a. Foreign bodies.

DISEASES OF THE GENERATIVE SYSTEM.

DISEASES OF THE MALE ORGANS OF GENERATION.

PENIS.

599. Inflammation.
600. Abscess.
(581^a.) *Gonorrhœa*.
601. Gangrene.
602. *Priapism.
(48^a.) *Syphilis*.
(49^a.) *Cancer*.
 a. Of prepuce.
 b. Of body.
603. Non-malignant tumors.
(1069.) *Injuries*.
604. Phimosiis—congenital.

SCROTUM.

605. Sloughing.

606. Edema.
607. Elephantiasis.
(828.) *Prurigo*.
(48^a.) *Syphilis*.
(49^a.) *Cancer*.
(49^c.) *Epithelial cancer*. *Syn.*, Chimney-sweeper's cancer.
608. Non-malignant tumor.

CORD.

609. Hydrocele.
 Varieties:
 a. Encysted.
 b. Diffused.
610. Varicocele.

* When the cause of this affection has been ascertained, the case should be returned under the head of the primary disease, the secondary affection being also specified.

† When the cause of the stricture is known it should be stated.

611. Non-malignant tumor.
612. Neuralgia.

TUNICA VAGINALIS.

613. Inflammation.
614. Hydrocele.
 Varieties:
 a. Congenital.
 b. Infantile.
 c. Encysted.
615. Hæmatocele.
616. Loose bodies.

TESTICLE.

617. Orchitis.

- a. Acute.
 b. Chronic.
618. Abscess.
619. Protrusion of tubuli. *Syn.*, Hernia testis. Fungus testis.
620. Atrophy.
 (48¹.) *Syphilitic disease.*
 (49¹.) *Cancer.*
621. Non-malignant tumor.
622. Cystic disease.
 (53¹.) *Tubercle.*
 (1069.) *Injuries.*
623. Fœtal remains in testicle.
624. Malposition.
625. Spermatorrhœa.
626. Impotence.
627. Neuralgia.

DISEASES OF THE FEMALE ORGANS OF GENERATION IN THE UNIMPREGNATED STATE.

Ovary.

628. Inflammation.
629. Abscess.
630. Hemorrhage.
631. Atrophy.
632. Hypertrophy.
 (49.) *Cancer.*
633. Fibrous tumor.
634. Encysted dropsy.
635. Complex cystic tumors. *Syn.*, Alveolar, gelatinous, and colloid tumor of the ovary. Cysto-sarcoma.
 a. With intracystic growths.
636. Cysts, containing tegumentary structures—hair, teeth, and bones.
 Syn., Dermoid cysts.
 (53¹.) *Tubercle.*
637. Cyst containing hydatid.
638. Dislocation.
639. Hernia.

FALLOPIAN TUBE.

640. Abscess.
641. Dropsy.
642. Stricture.
643. Occlusion.
 (49¹.) *Cancer.*
644. Cyst.
 (53¹.) *Tubercle.*
645. Dislocation.
646. Hernia.

BROAD LIGAMENT.

647. Inflammation.
 a. Pelvic peritonitis.
 b. Pelvic cellulitis.
648. Abscess.
649. Cyst.

- (817.) Peri-uterine or pelvic hæmatocele.

UTERUS.

650. Catarrh. *Syn.*, Leucorrhœa.
 a. Hydrorrhœa.
651. Inflammation.
 Granular inflammation.
 Abrasion.
652. Ulcer.
653. Rodent ulcer.
654. Abscess.
655. Utero-vesical fistula.
656. Stricture of the orifice.
657. " of the canal.
658. Occlusion of the orifice.
659. " of the canal.
660. Hypertrophy.
661. Atrophy.
 (49¹.) *Cancer.*
 a. *Scirrhus.*
 b. *Medullary cancer.*
 c. *Epithelial cancer.*
662. Fibrous tumor.
663. Polypus.*
 (53¹.) *Tubercle.*
664. Displacements and distortions.
 a. Antiversion.
 b. Retroversion.
 c. Antiflexion.
 d. Retroflexion.
 e. Inversion.
 f. Prolapsus.
 1. *Procidentia.*
 g. Hernia.

VAGINA.

665. Catarrh. *Syn.*, Leucorrhœa.
666. Inflammation.

* Under this head should be returned all pedunculated tumors growing from the cavity or neck of the uterus, whether mucous, cellular, or fibrous.

667. Abscess.
 (580^b.) *Gonorrhœa*.
 668. Cicatrix or band.
 668^a. Vaginal fistula.
 669. Vesico-vaginal fistula.
 670. Recto-vaginal fistula.
 671. Hernia.
 a. Cystocele.
 b. Rectocele.
 (49¹.) *Cancer*.
 672. Laceration.
 673. Polypus.

VULVA.

- 673^a. Inflammation of labia.
 674. Pruritus of labia.
 (853.) *Eczema of labia*.
 675. Œdema of labia.
 676. Abscess.
 677. Gangrene.
 678. Hypertrophy.*
 a. Elongation of cervix.
 679. Occlusion.
 680. Imperforate hymen.
 (262.) *Varicose veins*.
 (48¹.) *Syphilis*.

- (49¹.) *Cancer*.
 681. Vascular tumor of the meatus urinarius.
 682. Mucous cyst.
 (585^b.) *Condyloma*.

FUNCTIONAL DISEASES.

688. Amenorrhœa. *English Syn.*, Absent menstruation.
Varieties:
 a. From original defective formation.
 b. From want of development at the time of puberty.
 c. From mechanical obstruction.
 d. From temporary suppression.
 688. *Chlorosis*. *English Syn.*, *Green sickness*.
 684. Scanty menstruation. *Syn.*, Deficient menstruation.
 685. Vicarious menstruation.
 686. Dysmenorrhœa. *English Syn.*, Painful menstruation.
 687. Menorrhagia. *English Syn.*, Excessive menstruation.

AFFECTIONS CONNECTED WITH PREGNANCY.

†DISORDERS OF THE NERVOUS SYSTEM.

- Neuralgia.
Varieties:
 a. Odontalgia.
 b. Cephalalgia.
 c. Mastodynia.
 Chorea.
 Convulsions.
 Hypochondriasis.
 Mania.

†DISORDERS OF THE CIRCULATORY SYSTEM.

- Varicose veins—
 a. Of the lower extremities.
 b. Of the labia.
 c. Of the rectum. Hæmorrhoids.
 Serous exudation.
Varieties:
 a. Ascites.
 b. Œdema of labia.
 c. Œdema of lower extremities.
 Syncope.
 Palpitation.

†DISORDERS OF THE RESPIRATORY SYSTEM.

- Dyspnœa.
 Orthopnœa.
 Cough.

†DISORDERS OF THE DIGESTIVE SYSTEM.

- Salivation.
 Depraved and capricious appetite.

- Nausea and vomiting.
 Cardialgia or Heartburn.
 Pyrosis.
 Intestinal cramp—colic.
 Constipation.
 Diarrhœa.
 Jaundice.

†DISORDERS OF THE URINARY SYSTEM.

- Albuminuria.
 Dysuria.
 Incontinence of urine.
 Retention of urine.

DISORDERS OF THE GENERATIVE SYSTEM.

689. Metritis. *Syn.*, Hysteritis.
 690. Discharge of watery fluid from the uterus. Hydrorrhœa.
 691. Rheumatism of the uterus.
 692. Hysteralgia.
 693. Spurious pains and cramps.
 (665.) *Catarrh of vagina*. *Syn.*, *Leucorrhœa*.
 694. Sanguineous discharge. *Syn.*, Menstruation.
 695. Hemorrhage.
 696. Displacement of uterus.
Varieties:
 a. Prolapsus.
 b. Hernia.
 c. Retroversion.
 (674.) *Pruritus of the vulva*.
 697. Abortion.

* Specify the part.

† These affections are secondary, and are therefore not numbered.

698. Premature labor.
699. Extra-uterine gestation.

AFFECTIONS CONNECTED WITH PARTURITION.

700. Atony of the uterus.
701. Over-distension of the uterus.
 a. From excess of liquor amnii.
 b. " twins, triplets, &c.
702. Mechanical obstacle to the action of the uterus.
 a. From occlusion of the os uteri.
 b. From rigidity of the—
 os uteri.
 vagina.
 perineum.
 c. From cancer of the cervix uteri.
 d. " narrowness of the vagina.
 e. " cicatrix or band in the vagina.
 f. From vaginal cyst.
 g. " prolapsus of the bladder.
 h. " stone in the bladder.
 i. " distended rectum.
 k. " prolapsus of the rectum.
 l. " tumor.
 Varieties:
 1. Uterine.
 2. Ovarian.
 3. Pelvic.
 4. Of external parts.
 m. From polypus.
 n. " fractured pelvis.
 o. " exostosis.
 p. From distorted or contracted pelvis.
 q. From dislocated lumbar vertebrae into pelvis. *Syn.*, Spondylo listhesis.
 r. From ankylosed coccyx.
 s. From diminutive pelvis.
 t. Extreme anteversion of uterus (with pendulous abdomen).
 u. From excessive size of fœtus.
 v. " malposition of fœtus.
 w. " malformation of fœtus.
 z. From enlargement of fœtus from disease.
 y. From unusual thickness of foetal membranes.
 z. From unusual shortness of funis.
703. Hemorrhage.
 a. From placenta prævia. *Syn.*, Unavoidable hemorrhage.
 b. From accidental detachment of

- placenta. *Syn.*, Accidental hemorrhage.
 c. Thrombus of cervix uteri or labium.
704. Rupture or laceration of the—
 uterus.
705. vagina.
706. urinary bladder.
707. perineum.
708. Retention of the placenta.
 a. From atony of the uterus.
 b. From irregular or hour-glass contraction.
 c. From preternatural adhesions.
709. Inversion of the uterus.
710. Convulsions.

AFFECTIONS CONSEQUENT ON PARTURITION.

711. Post-partum hemorrhage.
(83.) *Puerperal ephemera*.
712. Milk fever.
(82.) *Puerperal fever*.
713. Metro-peritonitis. *Syn.*, Puerperal peritonitis.
 a. Metritis.
 b. Peritonitis.
(257.) *Phlebitis*.
(258.) *Phlegmasia dolens*.
714. Pelvic cellulitis.
715. Iliac and pelvic abscesses.
716. Sloughing of cervix uteri.
717. " vagina.
718. " perineum.
719. " bladder.
720. " rectum.
(655.) *Utero-vesical fistula*.
(669.) *Vesico-vaginal fistula*.
(670.) *Recto-vaginal fistula*.
(723.) *Mammary inflammation*.
(724.) *Mammary abscess*.
721. Puerperal mania.
 a. Connected with parturition.
 b. " " lactation.
722. Puerperal convulsions. *Syn.*, Eclampsia.
723. Sudden death after delivery.
 a. From shock or nervous exhaustion.
 b. From impaction of coagula in the heart and great vessels.
 1. Thrombosis.
 2. Embolism.
 c. From entrance of air into veins (from separation of placenta)
(896.) *Still-born*.
(897.) *Premature birth*.

DISEASES OF THE FEMALE BREAST.

- 723*. Inflammation.
 a. Acute.
 b. Chronic.
724. Abscess.
725. Sinus.
726. Galactorrhœa.
727. Deficiency of milk.

728. Hypertrophy.
729. Atrophy.
730. Depressed nipple.
731. Chapped nipple.
732. Ulcerated nipple.
(491.) *Cancer*.
 a. *Scirrhus*.

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|---|---|
| <p>b. <i>Medullary cancer.</i>
 c. <i>Epithelial cancer.</i>
 (50.) <i>Colloid.</i>
 733. Fibrous tumor. <i>Syn.</i>, Painful subcutaneous tumor.
 734. Fibro-plastic tumor.
 735. Fatty tumor.
 736. Osseous tumor.
 737. Cartilaginous tumor. <i>Syn.</i>, Enchondroma.</p> | <p>738. Chronic mammary tumor. <i>Syn.</i>, Adenoid tumor.
 739. Vascular tumor.
 740. Cyst.
 741. Complex cystic tumor. <i>Syn.</i>, Cystosarcoma.
 742. Hydatid.
 743. Hyperæsthesia.
 744. Mastodynia. <i>Syn.</i>, Neuralgia.</p> |
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DISEASES OF THE MALE MAMMILLA.

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| <p>745. Inflammation.
 746. Hypertrophy.
 (49¹.) <i>Cancer.</i></p> | <p>747. Non-malignant tumor.
 748. Cyst.</p> |
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DISEASES OF THE ORGANS OF LOCOMOTION.

DISEASES OF BONES.*

- | | |
|--|--|
| <p>749. Ostitis.
 a. Periostitis.
 1. Nodes.
 750. Diffuse periostitis. <i>Syn.</i>, Acute periosteal abscess.
 a. Acute necrosis.
 751. Osteo-mylitis.
 752. Chronic abscess.
 753. Caries.
 754. Necrosis.
 755. Mollities ossium.
 756. Hypertrophy.
 757. Atrophy.
 758. Spontaneous fracture. (The cause, if known, should be stated.)
 (48¹.) <i>Syphilitic disease.</i>
 (49¹.) <i>Cancer.</i>
 759. Non-malignant tumors.
 a. Fibrous and fibro-cystic.
 b. Myeloid.
 c. Cartilaginous. <i>Syn.</i>, Enchondroma.
 d. Exostosis.
 1. Diffused exostosis.
 760. Cyst.
 (54.) <i>Rickets.</i>
 (53.) <i>Scrofulous disease.</i>
 761. Hydatid.</p> | <p>767. Dropsy of joint.
 (36.) <i>Gonorrhæal rheumatism.</i>
 (37.) <i>Synovial rheumatism.</i>
 (42.) <i>Gouty synovitis.</i>
 (43.) <i>Chronic osteo-arthritis.</i> <i>Syn.</i>, <i>Chronic rheumatic arthritis.</i>
 768. Degeneration of cartilage and articular surfaces of bones.
 769. Perforation of joints.†
 770. Loose cartilage. <i>Syn.</i>, Loose body.
 771. Relaxation of ligaments.
 772. Displacement of articular cartilage.
 773. Knock-knee.
 774. Bow-leg, or out-knee.
 (49¹.) <i>Cancer.</i>
 775. Non-malignant tumor.
 776. Neuralgia of joints.</p> |
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DISEASES OF THE SPINE.

777. Ulceration of ligaments and cartilages.
778. Caries and necrosis.
a. Spontaneous fracture of odontoid process.
779. Psoas, lumbar, and other abscesses.
780. Angular deformity. *Syn.*, Kyphosis.
781. Lateral curvature. *Syn.*, Skoliosis.
782. Anterior curvature. *Syn.*, Lordosis.
(54.) *Rickety curvature.*
783. Ankylosis.
(43.) *Chronic osteo-arthritis.*
784. Non-malignant tumor.
(49¹.) *Cancer.*
785 Hydatid.
786. Deformity from malformations.

DISEASES OF THE MUSCULAR SYSTEM.‡
MUSCLE.

- | | |
|---|---|
| <p>762. Acute synovitis.
 763. Chronic synovitis.
 a. Pulpary degeneration of synovial membrane.
 b. <i>Strumous disease of joints.</i>
 1. <i>Morbus coxæ.</i>
 764. Ulceration of cartilage.
 765. Abscess.
 a. <i>Pyæmic abscess.</i>
 766. Ankylosis.
 a. Deformity from ankylosis.</p> | <p>787. Inflammation.
 788. Abscess.
 789. Gangrene.
 790. Atrophy.</p> |
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* In all cases the bone affected must be specified.

† In all cases the joint affected is to be specified.

‡ This refers to perforation by disease, and should be returned with the original affection.

§ In all cases the affected muscle or muscles should be stated.

791. Progressive atrophy.
Locomotor ataxy.
792. Fatty degeneration.
793. Ossification.
(48¹.) *Syphilitic deposit.*
(49¹.) *Cancer.*
(50¹.) *Colloid.*
794. Non-malignant tumor.
a. Erectile tumor.
795. Cyst.
Rupture.
(86.) *Infantile paralysis.*
796. Trichinosis.
(93.) *Spasm.*
797. Exhaustion.
(87^b.) *Scrivener's palsy.*
(19^a.) *Diphtheritic paralysis.*

TENDONS.

798. Inflammation.
(875^a.) *Thecal abscess.*
799. Adhesion of tendons.
(49¹.) *Cancer.*
800. Non-malignant tumors.

801. Contraction of tendons, fasciæ, or muscles.

802. Club-foot.

- a. *Talipes varus.*
b. " *valgus.*
c. " *equinus.*
d. " *calcaneus.*
e. " *calcaneo-varus.*
f. " *equino-valgus.*
Syn., Flat-foot.

803. Club-hand.

804. Contracted palmar fascia.

805. Wry neck.

(1135.) *Rupture of tendon.*

APPENDAGES OF MUSCULAR SYSTEM.

806. Enlarged bursa patellæ. *Syn., House-maid's knee.*
807. Enlargement of other bursa (specify which).
808. Bursal tumor.
809. Bursal abscess.
810. Bunion.
811. Ganglion.
a. Diffused palmar ganglion.

DISEASES OF THE CELLULAR TISSUE.

812. Inflammation.
813. Abscess.
844. Inflammatory induration in the newly born.
815. Slough.
(27^b.) *Phlegmonous erysipelas.*
(856.) *Carbuncle.* *Syn., Anthrax.*
816. Obesity.

817. Hemorrhage.
a. Pelvic hæmatocoele.
818. Non-malignant tumor.
(49¹.) *Cancer.*
819. Parasites.
(135¹.) *Foreign substances.*
820. Emphysema.

DISEASES OF THE CUTANEOUS SYSTEM.

DISEASES OF THE SKIN.*

- (27.) *Erysipelas.*
821. Erythema. This term includes
1. Erythema læve.
2. Erythema fugax. *Syn., E. volaticum.*
3. Erythema marginatum.
4. " papulatum.
5. " tuberculatum.
6. " nodosum.
822. Intertrigo.
823. Roseola. (This term includes
1. *Roseola æstiva.*
2. " *autumnalis.*
3. " *symptomatica.*
4. " *annulata.*)
824. Urticaria. *English Syn., Nettle rash.*
a. Urticaria acuta.
b. " chronica.
(Under one or other of these heads are included
1. Urticaria febrilis.
2. " evanida.
3. " perstans.

4. Urticaria conferta.
5. " subcutanea.
6. " tuberculata.)
825. Pellagra.
826. Acrodynia.
827. Asturian rose.
828. Prurigo.
829. Lichen. (This term includes
1. Lichen simplex.
2. " pilaris.
3. " circumscriptus.
4. " agrius.
5. " tropicus. *English Syn., Prickly heat.*)

(The so-called Lichen lividus is really a form of Purpura.)

830. Strophulus. *English Syn., Red gum.*
Tooth rash. (This term includes
1. Strophulus intertinctus.
2. " confertus.
3. " candidus.)
(Strophulus albidus is referred to Acne.
" violaticus to Erythema.)

* Where the disease is local its situation should be specified.

831. Pityriasis. (This term includes Pityriasis capitis. *English Syn.*, Dan-driff.)
(Pityriasis versicolor is referred to Parasitic affections as a *Synonym* of Tinea versicolor.)
832. Psoriasis. (This term includes Lepra.)
833. Psoriasis vulgaris. *Syn.*, Lepra vulgaris.
a. Psoriasis guttata.
b. " diffusa.
c. " gyrata.
d. " inveterata.
834. Miliaria.
a. Sudamina.
835. Herpes.
a. Herpes phlyctænodes.
b. " circinatus.
c. " iris.
d. " zoster. *English Syn.*, Shingles.
836. Pemphigus. *Syn.*, Pompholyx.
a. Pemphigus acutus.
b. " chronicus.
c. " solitarius.
837. Eczema.
a. Eczema simplex.
b. " rubrum.
c. " impetiginodes.
d. " chronicum.
838. Impetigo.
a. Impetigo sparsa.
b. " confluens.
1. Figurata.
2. Larvalis.
839. Rupia.
a. Rupia simplex.
b. " prominens.
c. " escharotica. *Syn.*, Pemphigus gangrænosus.
840. Ecthyma.
841. Acne.
a. Acne punctata.*
b. " indurata.
c. " rosacea.
d. " strophulosa. *Syn.*, Strophulus albidus.
842. Sycosis. *Syn.*, Mentagra.†
843. Stearrhœa.
a. Stearrhœa simplex.
b. " nigricans.
844. Ichthyosis.
a. Ichthyosis vera.
b. " cornea.
845. Xeroderma.
846. Leucoderma. (This term includes Vitiligo.)
847. Albinismus.
848. Canities.
849. Melasma.
(282.) *Melasma Addisoni.* *English Syn.*, Addison's disease. *Bronze skin.*
850. Lentigo and Ephelis. *English Syn.*, Freckles.
851. Chilblain.
852. Frostbite.
853. Ulcer.
854. Fissures.
(852.) *Cancerum oris.*
855. Boil.
856. Carbuncle. *Syn.*, Anthrax.
(26.) *Malignant pustule.*
857. Onychia.
858. Onychia maligna.
859. Whitlow.
a. Thecal abscess.
860. Gangrene.
861. Hypertrophy.
862. Corn.
863. Bunion.
864. Elephantiasis arabum. *Syn.*, Barbadoes leg; Elephas.
(51.) True leprosy. *Syn.*, El. Græcorum.
865. Atrophy.
a. Linear atrophy.
b. Alopecia.
c. Atrophy of nails.
(49.) *Cancer.*
866. Fibro-cellular tumor.
867. Fatty tumor.
(263.) *Nævus vascularis.*
868. Nævus. *Syn.*, Port wine stain.
869. Nævus pilaris. *Syn.*, Mole.
870. Sebaceous tumor.
a. Steatoma.
871. Cornua.
872. Molluscum.
873. Warts.
874. Condyloma.
a. Male.
b. Female.
875. Cheloid.
876. Frambœsia.
(51.) *Lupus.*
(53.) *Scrofulous disease.*
877. Ingrown nail.
878. Silver-stain.
(986.) *Burns and Scalds.*
(101.) *Hyperæsthesia.*
879. Pruritus.
- (102.) *Anæsthesia.*
880. Ephidrosis.
881. Anidrosis.

PARASITIC DISEASES OF THE SKIN.

882. Tinea tonsurans. *Syn.*, Ringworm.
Parasite, Trichophyton tonsurans.
883. Tinea decalvans. *Syn.*, Alopecia areata. *Par.*, Microsporon Audouini.
884. Tinea favosa. *Syn.*, Favus. *Par.*, Achlorion Schœnleinii, Pucciani Favi.
885. Tinea versicolor. *Syn.*, Pityriasis versicolor. *Par.*, Microsporon furfur.

* When the Demodex folliculorum is discovered, its presence should be stated.

† When the Microsporon mentagrophytes is discovered, its presence should be stated.

886. *Tinea Polonica*. *Syn.*, Plica Polonica. *Par.*, Trichophyton sporuloides.
887. *Mycetoma*. *Syn.*, Madurafoot. *Par.*, Chinonyphe Carteri.
888. *Scabies*. *Syn.*, Itch. *Par.*, Sarcoptes scabiei.
889. Phthiriasis, from Phthirius inguinalis. *English Syn.*, Crab-louse.
890. Irritation caused by
 a. *Pediculus capitis*.
 b. " *palpebrarum*.
 c. " *vestimentis*.
 d. " *tabescentium*.
891. *Pulex penetrans*. *English Syn.*, Chigoe.
- Pulex irritans*.
 892. *Cimex*.
 893. *Leptothrix autumnalis*. *English Syn.*, Harvest-bug.
 894. Wasps, bees, and other stinging insects.
 895. Nettles and other stinging plants.
- CONDITIONS NOT NECESSARILY ASSOCIATED WITH GENERAL OR LOCAL DISEASE.
896. Still-born.
 897. Premature birth.
 898. *Old age*.
 899. *Debility*.

POISONS.

In returning cases of poisoning, the precise agent should be stated.

METALS AND THEIR SALTS.

900. Arsenic.
 901. Mercury.
 (96.) *Mercurial tremor*.
 902. Lead.
 a. Lead colic. *Syn.*, Painters' colic.
 b. Lead palsy.
 c. Blue gum.
 d. *Metallic stain of conjunctiva (from lead)*.
 903. Copper.
 904. Antimony.
 905. Zinc.
 906. Silver.
 a. Silver-stain.
 907. Iron.
 908. Bismuth.
 909. Chromium.
 a. (Bichromate of potash.)

CAUSTIC ALKALIES.

910. Potash.
 911. Soda.
 912. Ammonia.
 913. Alkaline salts.

METALLOIDS.

914. Phosphorus.
 915. Iodine.

ACIDS.

916. Sulphuric acid.
 917. Nitric acid.
 918. Muriatic acid.
 919. Phosphorous acid.
 920. Oxalic acid.
 921. Tartaric acid.

VEGETABLE POISONS.

922. Savin.

923. Croton oil.
 924. Elaterium.
 925. Colchicum.
 926. Black hellebore.
 927. White hellebore.
 a. *Veratria*.
 928. Squill.
 929. Ergot of rye.
 (46.) a. *Ergotism*.
 930. Opium.
 931. Indian hemp. (*Cannabis indica*).
 932. Alcohol.
 (108.) a. *Delirium tremens*.
 933. Ether vapor.
 934. Chloroform vapor.
 935. Henbane. (*Hyoscyamus*).
 936. Deadly nightshade. (*Belladonna*).
 a. *Atropia*.
 937. Thorn apple. (*Stramonium*).
 938. Prussic acid.
 a. Oil of bitter almonds.
 b. Laurel water.
 939. Cyanide of potassium.
 940. Nitro-benzole.
 941. Wourali Curara. (*Strychnos toxifera*).
 942. Hemlock. *Conium*.
 943. Monkshood. (*Aconium*).
 a. *Aconita*.
 944. Foxglove. (*Digitalis*).
 a. *Digitalin*.
 945. Tobacco.
 a. *Nicotin*.
 950. Hemlock dropwort. (*Enanthe crocata*).
 946. *Nux vomica*.
 a. *Strychnia*.
 b. *Brucia*.
 947. *Upas tieute*. (*Strychnos tieute*).
 948. *Upas antiar*.

* This mode of return is only to be employed when the cause of death is not traceable to definite disease.

† When the cause of this affection has been ascertained, the case should be returned under the head of the primary disease, the secondary affection being also specified.

949. Calabar bean. (*Physostigma venenosum*.)
 951. Fool's parsley. (*Æthusa cynapium*.)
 952. Water hemlock. (*Cicuta virosa*.)
 953. Camphor.
 954. Cocculus Indicus.
 955. Darnel. (*Lolium temulentum*.)
 956. Indian tobacco. (*Lobelia inflata*.)
 957. Laburnum.
 958. Yew. (*Taxus baccata*.)
 959. Poisonous fungi.
 a. Mouldy bread.

ANIMAL POISONS.

960. Spanish fly. (*Cantharides*.)
 961. Decayed and diseased meat.
 962. Poisonous meat.
 a. Sausages.
 963. Poisonous cheese.
 964. Poisonous milk.
 965. Poisonous fish.
 a. Mussels.

GASEOUS POISONS.

966. Ammonia.
 967. Nitrous acid vapor.
 968. Chlorine.
 969. Carbonic acid.
 970. Carbonic oxide.
 971. Coal gas.

972. Cyanogen.
 973. Sulphuretted hydrogen.
 974. Putrid and morbid exhalations.
 975. Other noxious effluvia.

MECHANICAL IRRITANTS.

976. Pounded glass.
 977. Steel filings.

POISONED WOUNDS.

Varieties:

979. *a.* By venomous animals.
 1. Snakes.
 2. Scorpions.
 3. *Stinging insects*.
 (894.) *b.* By animals having infectious disease.
 (23.) *Glanders*.
 (24.) *Farcy*.
 (25.) *Equina mitis*.
 (26.) *Malignant pustule*.
 (89.) *Hydrophobia*.
 (2.) *Cow-pox*.
 980. *c.* By dead animal matter.
 981. *d.* By morbid secretions.
 982. *e.* By vegetable substances.
 983. 1. Poisoned arrows.
 Wourali.
 984. 2. Subcutaneous injection.*
 985. *f.* By mineral substances.

INJURIES.

GENERAL INJURIES.

986. Burns and scalds (including explosions).†
 987. Lightning stroke.
 988. Multiple injury. (The cause and extent to be stated.)
 989. Asphyxia.
 a. Drowning.

- b.* Hanging.
c. Strangling.
d. Overlying.
e. *Gaseous Poisons*.
 990. Privation.‡
 991. Exposure to cold.‡
 992. Infant exposure.‡
 993. Neglect.‡

LOCAL INJURIES.§

INJURIES OF THE HEAD AND FACE.

A.—OF THE HEAD.

994. Contusion.
 a. Cephalhæmatoma.
 995. Scalp-wound: bone not exposed.
 996. " bone exposed.
 997. Concussion of the brain.
 998. Fracture of the vault of the skull.
 a. Simple, without depression.
 b. " with depression.

- c.* Compound, without depression.||
d. " with depression.
 999. Hernia cerebri.
 1000. Fracture of the base of the skull.
 1001. Wound of the skull.||
 1002. Laceration of the brain, without fracture.
 1003. Injuries of the vessels. (Specify which.)
 1004. Injuries of the cerebral nerves.

* In returning such cases, specify the agent employed.

† When limited to one part of the body, the part is to be specified: *e. g.*, Scald of the larynx.

‡ Any affection that may have been induced by this cause ought to be stated.

§ In all cases of injury, specify whether accidental, judicial, homicidal, self-inflicted, or in battle.

|| In such cases state the main features of the case in the fewest words possible.

¶ If from gunshot, to be stated.

B.—OF THE FACE.

- 1005. Contusion.
- 1006. Wound.*
- 1007. Foreign bodies in the ear.
- 1008. " " nose.
- 1009. " " antrum.
- 1010. " " soft parts.
- 1011. Fracture of the facial bones.
- 1012. " lower jaw.†
- 1018. Dislocation of the jaw.

INJURIES OF THE EYE.

- 1014. Contusions.
- 1015. Contusions, with rupture of the sclerotic. *Syn.*, ruptured globe.
- 1016. Contusion, with dislocation of the lens.
- 1017. Contusion, with hemorrhage into the globe.
- 1018. Foreign bodies in the cornea or conjunctiva.
- 1019. Foreign bodies in the cavity of the eye.‡
- 1020. Wounds of the eyelids.
- 1021. " conjunctiva.
- 1022. " sclerotic.
- 1023. " cornea.
- 1024. " lens.
- 1025. " iris.
- 1026. Dislocation of the globe.
- (158.) *Total disorganization of the eye from injury.*
- 1027. Wounds and injuries of the parts within the orbit.
- 1028. Chemical injuries of the eyelids and eye.
- 1029. Burns and scalds.

INJURIES OF THE NECK.

- 1030. Contusion of the soft parts.
- 1031. Fracture of the hyoid bone.
- 1032. " cartilages of the larynx.
- 1033. Rupture of the trachea.
- 1034. Dislocation of the hyoid bone.
- 1035. Wound.
 - a. Superficial.
 - b. Cut throat.*
 - c. Gun-shot.*
 - d. Of great vessels.*
 - e. From the mouth.
- (986.) *Burn and scald of larynx.*
- 1036. Foreign bodies in the air-passages.
- 1037. " " pharynx.
- 1038. " " œsophagus.
- 1039. Injury of pharynx and œsophagus by corrosive substances.

INJURIES OF THE CHEST.*

- 1040. Contusion.
- 1041. Fracture of the ribs (including costal cartilages) without injury to lung.
- 1042. Fracture of the ribs (including costal cartilages) with injury to lung.
- 1043. Fracture of sternum.
- 1044. Wound of parietes.
- 1045. Perforating wound of chest.*‡
- 1046. Perforating wound of pleura or lung.*‡
- 1047. Wound of anterior mediastinum.*‡
- 1048. Wound of pericardium and heart.*‡
- 1049. Wound of vessels.*‡
- 1050. Rupture of heart or lung without wound or fracture.*

INJURIES OF THE BACK. (Including the whole spinal region.)

- 1051. Contusion.
- 1052. Sprain.
- 1053. Wound.*‡
- 1054. Fracture and dislocation of spine.||
- 1055. Injury of the cord without known fracture.

INJURIES OF THE ABDOMEN.

- 1056. Contusion.
- 1057. Contusion with rupture of muscles.*
- 1058. Contusion with rupture of viscera.
- 1059. Wound of parietes.‡
- 1060. Wound of parietes, with protrusion of uninjured viscera.
- 1061. Wound of parietes, with protrusion of wounded viscera.
- 1062. Wound of parietes with wound of unprotruded viscera.
- 1063. Wound of viscera without wound of parietes.*
- 1064. Foreign bodies in the peritoneal cavity.
- 1065. Foreign bodies in the stomach.
- 1066. Foreign bodies and concretions in the intestines.
- 1067. Fistula from injury, and artificial anus.

INJURIES OF THE PELVIS.

- 1068. Contusion.
- 1069. Wound of the male perineum, scrotum, and penis.‡
- 1070. Wound of the female perineum and vulva.
- 1071. Wound of the vagina and internal female organs.*

* In such cases, state the main features of the case in the fewest words possible.

† Injuries of the alveoli and teeth are to be returned with the other affections of those parts.

‡ If from gunshot, to be so stated.

§ Specify when from gunshot.

|| The seat of the injury, and the existence and extent of paralysis, to be stated.

1072. Wound of the rectum.*
 1073. " anus.
 1074. " bladder.
 1075. Rupture of bladder without wound.
 1076. Rupture of bladder from fracture.†
 1077. Injuries of the pregnant uterus.
 1078. Foreign bodies in vagina.
 1079. " " rectum.
 (566, 598.) " " bladder and urethra.‡
 1080. Fracture and dislocation of pelvis.
 1081. Fracture and dislocation of pelvis, with rupture of bladder or urethra.

INJURIES OF THE UPPER EXTREMITIES.

1082. Contusion.
 1083. Wound.*‡
 1084. Wound of joint.
 1085. " vessels.*
 1086. Sprain. (Specify which joint.)
 1087. Dislocation. (When compound, to be so stated.)
 1088. Dislocation of sterno-clavicular joint.
 1089. Dislocation of acromio-clavicular joint.
 1090. Dislocation of shoulder.
 1091. Dislocation of elbow.
 1092. " wrist and carpus.
 1093. " thumb.
 1094. " phalangeal joints.
 1095. Separation of epiphyses.
 1096. Foreign bodies embedded.*
 1097. Greenstick fracture, or bending of bone. (Specify which bone.)
 1098. Fracture. (State whether simple or compound.)
 1099. " of clavicle.
 1100. " scapula.
 1101. " humerus.
 1102. " forearm.
 1103. " carpus, metacarpus, and phalanges.
 1104. Ununited fracture, or false joint. (Specify which bone.)

INJURIES OF THE LOWER EXTREMITIES.

1105. Contusion.
 1106. Sprain. (Specify which joint.)
 1107. Wound.*‡
 1108. " of joint.
 1109. " of vessels.*
 1110. Foreign bodies embedded.*
 1111. Separation of epiphyses.
 Fracture. (When compound, to be so stated.)
 1112. " of femur.
 1113. " of cervix femoris.
 1114. " Intra capsular.
 1115. Fracture of trochanter major.
 1116. " patella.
 1117. " leg, both bones.
 1118. " tibia alone.
 1119. " " fibula alone.
 1120. " bones of the foot.
 Dislocation. (When compound, to be so stated.)
 1121. " of hip.
 1122. " patella.
 1123. " knee.
 1124. " head of fibula.
 1125. " foot, at the ankle.
 1126. " " at calcaneo-astragaloid, and scapho-astragaloid joints.
 1127. " astragalus.
 1128. " os calcis.
 1129. " other tarsal bones.
 1130. " metatarsus, and phalanges.
 1131. Ununited fracture, or false joint. (Specify which bone.)

INJURIES OF THE ABSORBENT SYSTEM.

1132. Foreign bodies and concretions.
 1133. Wounds of lymphatics.
 1134. Rupture of muscle.
 1135. " tendon.
 1136. Foreign substances in cellular tissue.

* In such cases, state the main features of the case in the fewest words possible.

† Rupture of bladder from accumulation of urine is usually from stricture, and must be returned under the appropriate heading.

‡ Return such cases with calculus in the bladder and urethra.

§ Specify when from gunshot.

HUMAN PARASITES.

The Parasites are to be returned under Local Diseases.

SUBDIVISIONS.

1. ENTOTZOA.
2. ECTOZOA.
3. ENTOPHYTA AND EPIPHYTA.

ENTOTZOA.

CLASSES.

- A. CÆLELMINTHA. *English Syn.*, Hollow worms.
- B. STERELMINTHA. *English Syn.*, Solid worms.
- C. ACCIDENTAL PARASITES.

CLASS A.—CÆLELMINTHA.

- Ascaris lumbricoides.* (Linnaeus.) *Habitat*, Intestines.
Ascaris mystax. (Rudolphi.) *Hab.*, Intestines.
Trichocephalus dispar. (Rudolphi.) *Hab.*, Intestines.
Trichina spiralis. (Owen.) *Hab.*, Muscles.
Filaria Medinensis. (Gmelin.) *English syn.*, Guinea-worm. *Hab.*, Skin and subcutaneous tissues.
Filaria oculi. (Nordmann.) *Syn.*, *Filaria lentis.* (Diesing.) *Hab.*, Eye.
Strongylus bronchialis. (Cobbold.) *Hab.*, Bronchial tubes.
Eustrongylus gigas. (Diesing.) *Hab.*, Kidney; intestines.
Sclerostoma duodenale. (Cobbold.) *Syn.*, *Anchylostomum duodenale.* *Hab.*, Duodenum.
Dactylius aculeatus. (Curling.) *Hab.*, Bladder.
Oxyuris vermicularis. (Bremser.) *English syn.*, Thread-worm. *Hab.*, Rectum.

CLASS B.—STERELMINTHA.

- Bothriocephalus latus.* (Bremser.) *Hab.*, Intestines.
Bothriocephalus cordatus. (Leuckart.) *Hab.*, Intestines.
Tænia solium. (Linnaeus.) *Hab.*, Intestines.
Cysticercus of Tænia solium. *Syn.*, *Cysticercus telus cellulosæ.*
Tænia mediocanellata. (Küchenmeister.) *Hab.*, Intestines.

- Tænia acanthotrias.* (Weinland.) *Hab.*, Intestines.
Tænia flavopuncta. (Weinland.) *Hab.*, Intestines.
Tænia nana. (Siebold.) *Hab.*, Intestines.
Tænia lophosoma. (Cobbold.) *Hab.*, Intestines.
Tænia elliptica. (Batsch.) *Hab.*, Intestines.
Cysticercus of the Tænia marginata. *Syn.*, *Cysticercus tenuicollis.*
Echinococcus hominis, or Hydatid of the Tænia echinococcus. (Siebold.)
Fasciola hepatica. (Linnaeus.) *Hab.*, Liver.
Distoma crassum. (Busk.) *Hab.*, Duodenum.
Distoma lanceolatum. (Mehlis.) *Hab.*, Hepatic duct; bowels.
Distoma ophthalmobium. (Diesing.) *Hab.*, Eye.
Distoma heterophyes. (Siebold.) *Hab.*, Small Intestines.
Bilharzia hæmatobia. (Cobbold.) *Hab.*, Portal and venous blood.
Tetrastoma renale. (Della Chiaje.) *Hab.*, Tubes of kidney.
Hexathyridium venarum. (Treutler.) *Hab.*, Venous blood.
Hexathyridium pinguiicola. (Treutler.) *Hab.*, Ovary.

CLASS C.—ACCIDENTAL PARASITES.

- Pentastoma denticulatum.* (Siebold.) *Hab.*, Liver; small intestines.
Pentastoma constrictum. *Hab.*, Liver.
Estrus hominis. (Say.) *English syn.*, Larva of the gad-fly. *Hab.*, Intestines.
Anthomyia canicularis. (A. Farre.) *Hab.*, Intestines.

ECTOZOA.

- Phthirus inguinalis.* (Leach.) *English syn.*, Crab-louse.
Pediculus capitis. (Nitzsch.)
Pediculus palpebrarum. (Le Jeune in Guillemeau.)
Pediculus vestimenti. (Nitzsch.) *English syn.*, Body-louse.
Pediculus tabescentium. (Burmeister.)
Sarcoptes scabiei. (Latreille.) *Syn.*, *Acarus.* *English syn.*, Itch-insect.*

* To be returned amongst the parasitic diseases of the skin.

Demodex folliculorum. (Owen.)
Pulex penetrans. (Gmelin.) *Syn.*, Chigoe.

ENTOPHYTA AND EPIPHYTA.

Leptothrix buccalis. (Wedl. Robin.)
English syn., Alga of mouth.
Oidium albicans. (Link.) *English syn.*,
 Thrush fungus. *Hab.*, Mouth in cases
 of thrush, and certain mucous and cu-
 taneous surfaces.
Sarcina ventriculi. (Goodsir.) *Hab.*,
 Stomach.
Torula cerevisiæ. (Turpin.) *Syn.*, *Cryp-*
tococcus cerevisiæ. (Kützing.) *Eng-*
lish syn., Yeast plant. *Hab.*, Stomach,
 bladder, &c.

Chionyphe Carteri. *Hab.*, Deep tissues,
 and bones of the hands and feet.
Achorion Schönleini. (Remak.) *Hab.*,
*Tinea favosa.**
Puccinia favi. (Ardsten.) *Hab.*, *Tinea*
*favosa.**
Achorion Lebertii. (Robin.) *Syn.*, *Tri-*
chophyton tonsurans. (Malmsten.)
Hab., *Tinea tonsurans.**
Microsporon Audouini. (Gruby.) *Hab.*,
*Tinea decalvans.**
Trichophyton sporuloides. (Von. Wal-
 ther.) *Hab.*, *Tinea polonica.*
Microsporon furfur. (Eichstädt.) *Hab.*,
*Tinea versicolor.**
Microsporon mentagrophytes. (Gruby.)
Hab., Follicles of hair in Sycosis or
*Mentagra.**

The foregoing list might be extended by the addition of various parasitic vegetations, which have been reported under the names of Algæ, Fungi, Mycodermis, Leptomiti, &c., but the characters or the existence of which are still the subject of inquiry.

CONGENITAL MALFORMATIONS OF THE FŒTUS.

MALFORMATIONS RESULTING FROM INCOMPLETE DEVELOPMENT OR GROWTH OF PARTS.

OF THE BODY GENERALLY.

Head absent, or rudimentary.
 Cranium defective.
 Lower jaw absent or defective.
 Upper and lower extremities absent.
 Lower extremities absent.
 One lower extremity absent.
 Hands and feet articulated to scapulæ and pelvis.
 Fingers and toes deficient in number.

OF THE NERVOUS SYSTEM.

Brain absent.
 Brain rudimentary or incompletely developed.
 Spinal cord absent or imperfect.
 Continuity of nerves with nerve-centres incomplete.

OF THE ORGANS OF SPECIAL SENSE.

Eyes absent.
 Eyes imperfect.
 Eyelids incomplete. Eyelids remaining united. (Symblepharon.)
 External ear absent. Pinna adherent.
 Meatus externus closed.
 Internal ear imperfect.
 Nose absent.

Nose imperfect.
 Nose resembling a proboscis.

OF THE VASCULAR SYSTEM.

Heart absent.
 Cavities of heart deficient in number.
 a. One auricle and one ventricle.
 b. Two auricles and one ventricle.
 Septa incomplete.
 a. Auricular.
 b. Ventricular.
 Orifices obstructed or imperfect.
 a. Right auricle, ventricular aperture.
 b. Pulmonic aperture.
 c. Left auriculo-ventricular aperture.
 d. Aortic aperture.
 Foramen ovale permanently closed.
 Orifices of aorta and pulmonary artery transferred.
 Orifices of ascending aorta from left ventricle and of descending aorta from right ventricle through the ductus arteriosus.
 Commencement of descending aorta contracted or obliterated.
 Foramen ovale persistent.
 Ductus arteriosus pervious.
 Cardiac valves imperfect.
 Pericardium absent.

OF THE RESPIRATORY SYSTEM.

Lung (one or both) absent.

* To be returned amongst the parasitic diseases of the skin.

Pulmonary lobes deficient in number.
Larynx and trachea absent or imperfect.

OF THE DIGESTIVE SYSTEM.

Esophagus impervious.
Intestine impervious, or deficient in various regions.
Anus impervious.
Anus in unusual situations.
Liver preternaturally small.
Gall-bladder absent.
Biliary ducts impervious.
Urachus patent. Vitelline duct patent.

OF THE URINARY SYSTEM.

Kidney (one or both) absent.
Kidney lobulated.
Ureters absent or impervious.
Urachus persistent.

OF THE MALE ORGANS OF GENERATION.

Penis diminutive, resembling clitoris.
Prepuce abbreviated—elongated.
Testis (one or both) absent.
External organs absent.

OF THE FEMALE ORGANS OF GENERATION.

Ovary (one or both) absent.
Uterus absent.
Vagina absent.
Vagina impervious.
Vagina a cul de sac.
External organs absent.

MALFORMATIONS RESULTING FROM INCOMPLETE COALESCENCE OF THE LATERAL HALVES OF PARTS WHICH SHOULD BECOME CONJOINED.

A.—ON THE ANTERIOR MEDIAN PLANE.

Fissure of the face.

“ iris. Coloboma.

“ lip.

a. Single harelip.

b. Double harelip.

Fissure of the palate.

a. Hard palate.

b. Soft palate.

“ nose. Naso-buccal fissure.

“ sternum.

“ diaphragm.

“ abdominal walls.

“ pubic symphysis.

“ anterior wall of urinary bladder (with extroversion of posterior half).

Epispadic fissure of the urethra.

Hypospadic fissure of the urethra.

Fissure of the scrotum.

B.—ON THE POSTERIOR MEDIAN PLANE.

Fissure of the skull.

“ spinal column.

Spina bifida.

a. Complete.

b. Partial.

1. Cervical region.

2. Lumbar “

3. Sacral “

Fissure of the spinal cord.

MALFORMATION RESULTING FROM COALESCENCE OF THE LATERAL HALVES OF PARTS WHICH SHOULD REMAIN DISTINCT.

Lower extremities conjoined. Syreniform fœtus.

Fingers or toes conjoined.

Monoculus. Cyclops.

Double kidney.

MALFORMATIONS RESULTING FROM THE EXTENSION OF A COMMISSURE BETWEEN THE LATERAL HALVES OF PARTS (CAUSING APPARENT DUPLICATION.)

Double uterus.

Double vagina.

MALFORMATIONS RESULTING FROM REPETITION OR DUPLICATION OF PARTS IN A SINGLE FŒTUS.

Supernumerary fingers and toes.

“ cavities to heart.

“ valves.

MALFORMATIONS RESULTING FROM THE COALESCENCE OF TWO FŒTUSES, OR OF THEIR PARTS.

Fœtus, more or less perfect, contained within another fœtus.

Fœtus, more or less perfect, constituting a tumor covered by integument.

Double fœtus.

a. One perfect. The other an appendage.

b. Both more or less perfect.

1. The middle parts united. The upper and lower distinct.

2. The upper parts united. The lower distinct.

3. The lower parts united. The upper distinct.

CONGENITAL DISPLACEMENTS AND UNUSUAL POSITIONS OF PARTS OF THE FŒTUS.

Transposition of viscera.

Hernia or ectopia of the—

brain.

heart.

Hernia or ectopia of the— lungs. intestines.	DISEASES MANIFESTED AT OR AFTER BIRTH.
<i>Varieties:</i> Through diaphragm. <i>Syn.</i> , Diaphragmatic hernia. Through abdominal walls. <i>Syn.</i> , Abdominal hernia. Through umbilicus. <i>Syn.</i> , Umbilical hernia. Extroversion of posterior wall of bladder. Testis retained in abdomen. Testis retained in inguinal canal.	Prematurely born. Stillborn—Asphyxia. Atelectasis pulmonum. Jaundice. Idiotcy. Dumbness or deaf-dumbness. Congenital cataract. Cephalhæmatoma. Syphilis.

PART III.

THE NATURE OF DISEASES—SPECIAL PATHOLOGY AND THERAPEUTICS.

It is the object of this part to treat of diseases in groups or classes, which possess certain characters or types common to the diseases composing each group; to describe, *Firstly*, The common properties or characters peculiar to the respective classes mentioned in the previous part on systematic medicine; to describe, *Secondly*, The several orders into which these classes of diseases may be subdivided; and, *Thirdly*, To describe in detail the several diseases individually, their *general nature and causes*; *symptoms, course, and complication*; *diagnosis, prognosis, and treatment*.

CLASS I.

ZYMOTIC DISEASES.

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CHAPTER I.

GENERAL REMARKS ON THE PATHOLOGY OF ZYMOTIC DISEASES.

THIS class comprises diseases which have been observed to be *epidemic, endemic, and contagious*, and includes *specific fevers, small-pox, plague, influenza, cholera*, and such other diseases as possess the peculiar character in common of suddenly attacking great numbers of people, at intervals, in unfavorable sanitary conditions. In the language of Dr. Farr, the "diseases of this class distinguish one country from another,—one year from another; they have formed epochs in chronology; and, as Niebuhr has shown, have influenced not only the fall of cities, such as Athens and Florence, but of empires; they decimate armies, disable fleets; they take the lives of criminals that justice has not condemned; they redouble the dangers of crowded hospitals; they infest the habitations of the poor, and strike the artisan in his strength down from comfort into helpless poverty; they carry away the infant from the mother's breast, and the old man at the end of life; but their direst eruptions are excessively fatal to men in the prime and vigor of age. They are emphatically the *morbi populares*."

The name *Zymotic* (first suggested by Dr. William Farr to desig-

nate the class) is not to be understood as implying the hypothesis that these diseases are fermentations, which the derivation of the term would lead one to believe. It has become extensively used of late as applied to the diseases whose characters as a class are already indicated, and for which some convenient term is required. The class, then, to which the term ZYMOTIC has been applied is intended to comprehend all the principal diseases which have prevailed as *epidemics* or *endemics*—all those which are due to *paludal* or *animal malaria*; and those due to *specific disease poisons*, capable of propagation from one human being to another, and *communicable either by direct contact*, or indirectly through various channels of human intercourse, contaminating drinking-water or infecting the air, or *by animals in a state of disease*. The class also comprehends the diseases that result from the *scarcity and the deterioration of the necessary kinds of food*, or from the generation, propagation, or existence of *parasitic animals*. The diseases of this class are thus conveniently arranged into four orders or groups, of which *specific fevers*, *syphilis*, *scurvy*, *worms*, are the common names typical of diseases in their respective groups.

In the greater number of the diseases of this class the blood is more or less changed, and by some is presumed to be the primary seat of diseases which result from specific poisons, of organic origin, either derived from without or generated within the body. These specific poisons tend to produce in the blood an excess of those decomposing organic compounds which physiology teaches us are always present in the circulating current.

The Physiological Modes in which Poisons act Illustrate by Analogy the Zymotic Diseases.—If the reader will now consider the following statements as to the modes in which poisons act physiologically, he will be prepared to appreciate the effects of those conditions which, like poisons, induce diseases of the class termed Zymotic. The actions of poisons are subject to certain general laws,—the most important of which are, *first*, that they have all certain definite and specific actions; *second*, that they all lie latent in the system a certain but varying period of time before those actions are set up; and *third*, that the phenomena resulting from their action vary, in some degree, according to the dose and to the receptivity of the patient. These laws are common to all poisons, but some are peculiar to individual poisons or classes of poisons, and it may be necessary to notice a few of them.

The *first* law, or that of the definite and specific actions of poisons, cannot be doubted: for if it be supposed that agents acting on the human body do not produce their effects according to certain definite laws, we can neither determine the seat nor the course of any disease, nor direct nor judge of the operation of remedies. No one, for instance, has seen castor oil produce tetanus, or colchicum intoxicate the brain, or opium inflame the spleen. The physician perfectly well knows that the first of these substances acts on the intestines, the second on the ligaments, and the third on the nervous system generally. The action of poisons, therefore, is not accidental, but determined by certain definite laws.

The action of poisons, though definite, is variously limited. Some poisons, for instance, act on one membrane, or on one organ, or on one system of organs; while other poisons extend their action over two or more membranes, or organs, or systems of organs, or even over the whole animal frame. We have examples in aloes and jalap of substances that act mainly upon the mucous membrane of the alimentary canal. In digitalis we have an instance of a medicine that principally acts on the heart, greatly reducing or even stopping its action; while strychnine is an example of a medicine acting on the parts supplied by the spinal cord, producing powerful and sometimes fatal tetanic action of every voluntary muscle in the body.

It is seldom, however, that the action of poisons is limited to one membrane, or organ, or system of organs. The greater number of these noxious agents more usually act on two or more membranes, or organs, or systems of organs. Elaterium, for instance, acts on the mucous membrane of the intestinal canal, and on the kidneys. Tobacco nauseates the stomach, intoxicates the brain, and affects the action of the heart. Antimony has an equally extensive range: it induces cutaneous perspiration, acts cathartically and emetically, and in large doses appears to cause gangrene of the lungs. Alcohol and opium are examples of substances acting still more generally, affecting not only the action or secretion of every organ or tissue of the body, but even in some instances altering their structure. Thus alcohol, in its most limited action, has been shown to cause structural disease of the liver, of the stomach, and of the coats of the arteries. From the circumstance of these substances acting not only generally, but inducing local lesion, they resemble in their effects those of many morbid poisons, as that of typhus fever, of scarlet fever, of small-pox, or of syphilis.

The *second* important law of poisons is, that they lie latent in the system for a period of time which varies in different individuals, before they set up their specific actions. Rhubarb, for instance, produces no immediate result, but lies dormant in the system six or eight hours before its action is sensible on the bowels; opium, in the usual dose, is generally thirty minutes before it subdues the brain. The convulsions from strychnine do not follow till twenty minutes after its administration; and perhaps every substance, except hydrocyanic acid, has a greater or less sensible period of latency.

When a medicine acts on more parts than one, a considerable space of time may elapse after it has affected one organ before it affects another: thus digitalis frequently occasions emesis before it acts on the heart, and the action of mercury on the bowels is frequently sensible for many weeks before the gums and salivary glands are affected. The doctrine of the latency of poisons is indeed so generally admitted that the actual period has been a point on which the condemnation or acquittal of a prisoner tried for murder has turned in our courts of justice, when certain poisons have been supposed to have been given.

The *third* great law of poisons is, that their effects are modified by the dose, the temperament, and the existing state of the consti-

tution, mentally and bodily, of the recipient. The effect of the dose in modifying the pathological phenomena of disease may be exemplified in the actions of oxalic acid and of arsenic. The specific action of oxalic acid is to inflame the mucous membrane of the stomach; but to insure this effect the dose must be limited so that this poison may lie in the system many hours. On the contrary, if the dose be excessive, and rapidly absorbed, the poison so disorders all the functions of the three great nervous centres that life is destroyed in a few minutes. Arsenic, likewise, is a poison which inflames and ulcerates the mucous membrane of the alimentary canal, but it requires some hours to set up its specific actions; for, when the dose is large, it, in like manner, destroys by general irritation, and before traces of morbid change of structure can be appreciated after death. It follows from this law, that the larger the dose, or the greater the intensity of the poison, the more rapid its action, and the less the probability of finding any trace of specific lesion induced by it.

In studying the effects of dose on the constitution, we find some poisons are absorbed and are *cumulative*, while others are not absorbed into the system; or they are so rapidly removed that no cumulative effect is produced. Thus, in persons predisposed to the effects of digitalis, a dose so small as to produce no sensible effect whatever, will, if frequently repeated, at last destroy the heart's action. This cumulative property of poisons, however, is by no means universal. There is no instance of jalap or of castor oil proving cumulative; and if a frequent repetition of either of them produces an increased effect, it is, perhaps, in consequence of the nervous papillæ with which they are brought in contact being more easily irritated by each application, and hence they induce a more violent result. That the habitual ingestion of decomposing matter in the water used as drink is capable of inducing conditions favorable to the development of Zymotic diseases, admits of no doubt. Cogent instances of this are to be found recorded in the bitter experience of epidemics of cholera.

Temperament is a circumstance which greatly influences the action of poisons. There are a few persons—rare exceptions—altogether insensible to the action of mercury, so that no quantity will affect their gums, or increase the secretion of the salivary glands. There are others, in like manner, the action of whose heart no quantity of digitalis will control. On the contrary, there are some constitutions—and these not so rare—so morbidly susceptible of these remedies that it is scarcely possible to administer even a fractional dose of these drugs without giving rise to their specific effects.

Besides natural temperament, *habit*, which may be termed an artificial temperament, has a powerful influence in reconciling us to particular classes of poisons, and of making them even sources of enjoyment. Thus tobacco, alcohol, opium, are all substances which are productive, in the first instance, to many persons, of great discomfort; but by frequent repetition they cease to have any unpleasant effects, and their stimulus at length becomes a necessary indulgence. Still there are many poisons to which no repetition can

habituate us. On the contrary, each repetition only the more debilitates the constitution, and renders it more susceptible of the action of the poison.

A peculiar existing state of the constitution has also a powerful influence on the action of poisons; and it would seem proved, with some exceptions, that these agents act with an intensity proportioned to the debilitated state of the patient. There is indeed no duty more imperative on the physician than that of adjusting the dose to the strength of the patient; and nothing is more common than to forbear administering a medicine because the patient's strength will not admit of it. As a general principle, therefore, medicines or poisons may be said to act with a power proportionate to the debility of the patient.

Still there are states of disease which render the constitution of the patient, though greatly debilitated, insusceptible to the action of even powerful remedies. Thus, in typhus fever, the patient will often bear a considerable quantity of vinous stimuli without being affected by it. In tetanus, or hydrophobia, no quantity of opium will tranquillize the symptoms or procure sleep. Fallopius mentions a singular instance of the constitution being armed against the action of a poison. He states that in his day a criminal was given up to himself and other anatomists, to be put to death in any manner they might think proper. To this man, therefore, they administered two drachms of opium, but, laboring under a quartan ague, and the fit just coming on, the "opium was hindered of its effect." The man, therefore, having survived this dose, begged that he might take a similar quantity, earnestly entreating, if he escaped, that he might be pardoned. The same dose was repeated, but it was in the interval of the attacks, and the man died.

The experiments of Majendie may be referred to as affording many curious proofs of the state of the constitution in accelerating or retarding the action of poisons. He has shown that if a poison be introduced into the system, of such potency as usually will destroy life in two minutes, on bleeding the animal the same result will follow in half a minute, or in one-fourth of the time; and this experiment has often been repeated. Majendie also brought to light the curious fact, that if, after having poisoned the animal, and even after the poison has begun to act, we inject an aqueous fluid into its veins in such quantity as to cause an artificial plethora, as long as this artificial plethora can be maintained, the action of the poison is superseded. No sooner, however, does the plethora cease, from the general effusion of fluid into every cavity of the body, than the poison acts in the usual time, and with even perhaps more than its accustomed severity.

Mr. Hunter thought that no two poisons could *coexist* in the same system together, or that, coexisting, they could not set up their specific actions at the same time. This hypothesis, however, is unquestionably erroneous; for we constantly see opium and digitalis, jalap and mercury, as well as many other combinations of medicines, producing their respective effects in the same system, and at the same time, by accelerating or retarding each other's

actions. There is no truth better established in medicine than that a combination of salts and senna produces a much more efficient and pleasant action than the administration of either remedy separately; and opium is an agent possessing a modifying or controlling power over every organ or tissue, without which it would be impossible, on many occasions, to reconcile the system to the introduction of many necessary and essential remedies. Poisons, therefore, are capable of coexisting together, and of so influencing the system that they reciprocally accelerate or retard each other's actions. The coexistence of two or more specific diseases has been already noticed at page 139.

The general laws observable in the actions of medicinal substances are for the most part precisely similar to those which govern morbid poisons, or only differ in a few minor points; for these latter poisons have their specific actions and their periods of latency, while their phenomena are not less variable, although the conditions of their varied actions are not yet clearly determined.

The Specific Action of Poisons which Produce Zymotic Diseases is distinctly proved by the fact that we are enabled to determine, within certain limits, the course, symptoms, and pathological phenomena which result from the presence of any given morbid poison. No man, for instance, can confound the phenomena of *small-pox* with those of *intermittent fever*, or those of *intermittent fever* with *syphilis*, or those of *syphilis* with *cholera*: each of these poisons has its separate and peculiar origin, course, development, and mode of propagation, and consequently their actions are so far definite and specific.

The actions of morbid poisons, like those of medicinal substances, are variously limited, some affecting only one membrane, or organ, or system of organs. Thus, *tinea* is an example of a noxious germ acting on one tissue of the body, and even then partially. In some parts of the world, for instance, in Switzerland, in the Brazils, in the Andes, and some of the Northwest provinces of India, a poison exists, associated with limestone and sometimes magnesian geological formation, whose action is limited to the undue ossification and thickening of the base of the cranium, tending to diminish the size of the foramina for the bloodvessels, and so leading to cretinism, and to growth of the thyroid gland in goitre (KÖLLIKER and REVIEWER in *B. and F. Med.-Chir. Review*, 1861, p. 43). Mr. Ceely, mentioning the fact that at Aylesbury, where goitre prevails, the soil is mainly limestone, incidentally states that solid aggregations of calcareous particles are also found in the thyroid gland. The contagion of *whooping-cough* and the virus of *hydrophobia* affect all the organs supplied by the eighth pair, or pneumogastric system. Instances of morbid poisons acting on several membranes, or organs, or systems of organs, are still more common, and form the great body of this class of diseases. The poison of *measles*, for instance, expresses itself no less on the mucous membrane of the eyes, nose, fauces, and perhaps on the mucous membranes generally, than on the skin; that of *scarlatina* not only on the mucous membrane of the fauces, and on the skin and the kidneys, but also on the serous

membranes of the joints and the abdomen. The *paludal* and the *syphilitic* poisons have a still more extensive range, hardly any organ or tissue of the body being exempt from the destructive ravages of these poisons.

Morbid, like other poisons, have their periods of latency; and, generally speaking, a much longer time elapses before their specific actions come into operation than with medicinal substances. The virus of the *natural small-pox* lies dormant from sixteen to twenty days before it produces any constitutional disturbance; and a still further period elapses, of three or four days, before the specific eruption appears on the skin. The poison of *scarlatina* lies latent from seven to ten days after exposure to the contagion; that of *measles* from ten to fourteen; while the poison of *paludal fever* has been said to lie dormant for a twelvemonth, and that of *hydrophobia* for a still longer time. These are examples of periods of latency far beyond anything that has been observed in the action of medicinal substances; and *syphilis* in its remote effects upon the organs and the constitution generally is still more remarkable.

When morbid poisons act on more tissues or organs than one, their actions are sometimes simultaneous, but more commonly they are consecutive, and frequently long intervals of time elapse between each successive attack. Thus, the poison of *typhus* and *enteric fever* may affect the lungs, the membranes of the brain, and the mucous membrane of the alimentary canal, and all these may be attacked contemporaneously, but more often consecutively; or first on the alimentary canal, then on the brain, and lastly on the lungs, several days elapsing between each successive affection.

It occasionally happens that morbid poisons which usually act on a plurality of membranes, exhaust themselves on one or more without affecting others. In *scarlatina simplex* the poison sometimes exhausts itself entirely on the skin, without affecting either the mucous or serous membranes of the body. The *rubeola sine catarrho* is a similar example of the poison exhausting itself on the skin. In *intermittent fever*, when the dose of the poison is limited, and the disease properly treated, it is seldom that a lesion occurs in any organ or tissue; yet, left to run a slow course, with constant exposure to the poison, scarcely any organ or tissue would escape being affected and its function impaired.

Sometimes, when the morbid poison acts on many membranes, the usual order of attack is inverted. In *scarlet fever* the affection of the skin may precede that of the throat, or the reverse may take place.

It has been seen that the period of latency of medicinal substances having passed over, the effects vary in a considerable degree, according to the dose, temperament, or present state of the constitution of the patient. With respect to the dose of a morbid poison, we rarely possess any direct measure of its strength. The *paludal poison* of tropical climates, to which *malarious fevers* are due, unquestionably greatly exceeds in intensity that of more temperate climates, and its effects are proportionally marked. Thus, in the West Indies, the severe remittent fevers occur with hardly a trace

of organic lesion after death, so rapid is their course; in Holland, a paludal fever of less severity exists, but it is followed by enlarged livers or spleens, or by dropsy; while, in this country, the same fever is comparatively mild, and if properly treated, for the most part terminates without any visceral affection. With respect to the influence of temperament in modifying disease, *small-pox* offers very striking instances. Different persons inoculated or poisoned from the same source have suffered in every degree from this formidable malady—from the *horn*, the *distinct*, the *confluent*, and the *bloody small-pox*; while, in the worst cases, children have died in the primary fever, and even before the specific action on the skin had time to be developed. It may therefore be laid down as a general law, that the more intense the dose of the morbid poison, the more severe the form and rapid the course of disease; and that fewer traces of organic alteration will be found after death when the poison is severe and abundant, than when the poison, or the disorder it produces, has been of a milder character, and the course of the disease more prolonged. Thus, enlarged livers, disorganized spleens, and dropsy, marked every case that died of the Walcheren fever; while in the West Indian and African fevers, though resulting from the same poison, scarcely a trace of disease was to be found.

The existing state of the constitution also influences the event. Thus persons of a good constitution, but ignorant of their danger, are often seen to pass through a mild form of typhus fever, while the nurses and others contaminated by the same poison, but more alive to their critical state, have sunk in a short time. A presentiment of death is a very unfavorable circumstance in the progress of *remittent* fever, especially in tropical climates. A soldier will sometimes say to the medical officer, "You have been very kind to me, sir; but this time I shall not get over it." There may be no appearance of absolute or immediate danger at the time—yet the man generally dies (SIR RANALD MARTIN). As a general principle, therefore, it may be stated that morbid poisons act with an intensity proportioned to the enfeebled or depressed state of the constitution; but this law is not universal. Want of a sufficient amount of food is most powerful among the conditions which predispose or help to bring about Zymotic diseases, and most constant in operation. It is a popular belief that the lowering of all the vital forces by deficiency of food constitutes the particular condition which renders a starved population so peculiarly open to the invasion of these diseases; but it is also a curious phenomenon of starvation that a state of general putrescence supervenes during life, as if the want of material for the generation of new tissue were an obstacle to the deportation of that which has become effete (CARPENTER). The hardy mountaineer is a surer victim, whether he visits the low countries of the tropics or the marshes of a more temperate climate, than the feeble native of those countries. The immunity the latter enjoys is probably owing to his habit of living in the noxious atmosphere; for let him remove to a more healthy climate, and then return to those regions of pestilence, and he will be found as susceptible of the poison as the hardier stranger.

Peculiarities in the action of Poisons which induce Zymotic Diseases.

—The principal points in which the effects of poisons which induce Zymotic diseases agree with those of poisons generally having been stated, it will now be necessary to state those circumstances in which they principally differ. Many medicinal poisons have the property of accumulating in the system, and acting with an intensity proportioned, not to the last dose, but to the aggregate of the whole quantity that has been administered. Thus the last few minims of digitalis may stop the action of the heart, or the last few grains of mercury salivate the patient, or the last minute dose of strychnine become fatal. There is, however, no well-authenticated fact which can be arranged under this law in the whole circle of morbid poisons, except, perhaps, the cumulative and persistent pernicious action of paludal malaria. The actual quantity required to establish disease, according to the experiments of Dr. Fordyce, is probably extremely small. That physician, in the hopes of mitigating the small-pox, inoculated with virus greatly diluted; and although the disease was not always produced, yet when produced, it assumed every form, character, and degree of severity that small-pox has ever been known to assume.

The puerperal female is not only highly susceptible of poisons of the Zymotic kind, but she is proved to favor their further development; and forms of puerperal fever seem capable of generation by *materies morbi* of a kind other than that which might be considered peculiar to it. It is a well-known fact, unhappily not of rare occurrence, that a medical practitioner or a nurse from a case of puerperal fever going to attend on other cases of labor, the chances are that these will be attacked with the disease. Further, the practitioner or nurse may go to cases of labor from attendance on a case of scarlatina, typhus, erysipelas, or small-pox, and the parturient patients may then become the victims of puerperal fever. In the Vienna Lying-in Hospital it is on record that a mortality of 400 to 500 in an average of 3000 deliveries per annum appeared traceable to the introduction of cadaveric matters, through the uncleanness of the attending students; these matters, being especially potent when derived from the bodies of those who have died from the adynamic forms of Zymotic disease. Students of practical midwifery should bear in mind this fact. They ought not to attend cases of labor while they are also engaged with practical anatomy in the dissecting-room.

Another peculiar law of morbid poisons, and one wholly unknown in medicinal substances, is the faculty which the human body possesses of generating to an immense extent a poison of the same nature as that by which the disease was originally produced. A quantity of small-pox matter not so big as a pin's head will produce many thousand pustules, each containing fifty times as much pestilent matter as was originally inserted; and, moreover, the blood and all the secretions of the body are equally infected with the matter of the pustules. The miasmata from one child laboring under whooping-cough are sufficient to infect a whole city.

There is still perhaps a more remarkable law of morbid poisons,

which is, that many of them possess the extraordinary property of exhausting the constitution of all susceptibility to a second action of the same poison. This is the case with syphilis, scarlatina, measles, typhus fever, small-pox, whooping-cough, and, indeed, with a considerable number of others. Still it would seem that a temporary protective influence is imparted by most morbid poisons, for it is certain that few persons suffer a second attack of the same specific epidemic disease; and, consequently, it follows that the previous action of the poison must for a time impair the susceptibility of the constitution to its attacks. This beneficent law is of great importance in social life; it enables those that have recovered to attend on those that are sick, and allows a mother fearlessly to nurse her child in a dangerous and contagious distemper she has herself passed through, if such an inducement is ever necessary to strengthen the moral courage of a mother.

The laws of poisons are not more important than their *modus operandi*; and this part of the subject has been deeply investigated by modern physiologists, and deserves some consideration. The great and striking alterations which often take place in the blood, led from a very remote period to the doctrine of humoralism, or, that a morbid state of the fluids was the great and primary cause of disease. On the contrary, when anatomy began to be cultivated, and nerves traced into every organ and tissue, it was supposed that disordered actions of these prime agents of motion, and of the great phenomena of animal life, were the great causes of disease, the morbid state of the fluids being secondary. Fontana, attempting to prove this latter theory, found, to his surprise, on laying bare the sciatic nerve in a great number of rabbits, that neither the venom of the viper nor hydrocyanic acid, when applied to it, produced the phenomena of poisoning, and that no other consequence resulted beyond what would have been produced by a similar mechanical injury. Having thus shown that the phenomena of poisoning do not result from the application of the deleterious agent to the trunk of the nerve or to the *solids*, he determined to ascertain whether they followed after absorption, and consequent contamination of the *fluids*. He injected the venom of the viper, hydrocyanic acid, and other poisonous substances directly into the veins of different animals; and he found that although the nerves of a part may be steeped in these poisons with impunity, yet no sooner did the substance enter the veins than the animal, after uttering a few horrible shrieks, struggled and almost instantly died, and thus demonstrated a morbid state of the fluids, as well as the existence of a tissue of extreme sensibility, with which the poison being brought into contact, accounted for the death of the animal. Fontana pursued this subject one step further, and showed, if poisons acted by absorption, that this absorption was in many instances extremely rapid. He submitted a number of pigeons to be bitten in the leg by a viper. He then chopped the wounded limb off at different intervals after the introduction of the venom, and found, as the result of an extensive series of experiments on several dozens of pigeons, that none recovered when the poisoned leg was removed at a later

period than twenty-five seconds, though the phenomena of poisoning did not occur till several minutes later.

The experiments of Fontana had shown (supposing a poison to be introduced into the veins) that all the phenomena of poisoning were accounted for; but still it might be said that to prove the fact of absorption something was wanting in strict demonstration. For the further prosecution of this subject we are indebted to Segalas, who showed that if the arteries and veins of the mesentery of a dog be tied, a quick-acting poison would lie in harmless contact with the corresponding portion of the intestine for many hours; but no sooner were these ligatures removed than poisoning took place in a few minutes. Majendie has carried this proof, of the veins absorbing, even still further. He amputated the leg of a dog, having first introduced a portion of quill into the femoral artery and vein, in such a manner that, on dividing these vessels, the leg hung connected with the trunk solely by means of the quill, all continuity by means of the solids being cut off. The poison was now introduced into the tissues of the paw, and in four minutes the animal was under its influence.

By these experiments it is believed that Fontana, Segalas, and Majendie have completely demonstrated the absorption of poisons by the veins, and consequently of their circulating with the blood; and that no doubt may remain on the subject, modern chemistry has demonstrated the actual presence of many medicinal substances either in the blood itself or in the secretions formed from it. Thus, after the free use of soda, large quantities of uncombined alkali have been found in the serum. Alcohol has been obtained by distillation from the blood; while iodine, rhubarb, the nitrate of potash, and a large number of other substances taken into the stomach have been found in the urine. It follows, then, that poisons are absorbed and mingled with the blood, and are conveyed directly to the parts on which they act, passing with impunity over others for which they have no affinity.

The fact of morbid poisons in like manner being absorbed, and mingling with the blood, has been shown by many Continental writers; but perhaps the experiment made by Professor Coleman is the most satisfactory. "I have produced the disease (the glanders) by first removing the healthy blood from an ass, until the animal was nearly exhausted, and then transfusing from a glandered horse blood from the carotid artery into the jugular vein. The glanders in the ass was rapid in its progress, violent in degree, and from this animal I afterwards produced both glanders and farcy." Scarlatina, measles, and syphilis have now been produced by inoculation from the blood of patients laboring under these diseases.

The circumstance of the presence of a poison in the blood is supposed by Andral to produce, besides its toxic states, certain alterations in its physical condition. Thus a specific cause has a tendency to destroy or reduce the quantity of fibrine in the blood, which he has found in some instances to be only one part in a thousand. Hence he adds, whatever may be the nature of the specific pyrexia, the blood always exhibits the following characters, whether it be

taken from a vein or collected from the heart and arteries after death,—namely, that the serum and clot are incompletely separated the one from the other, so that the clot is consequently large, and often appears to fill almost entirely the bleeding-basin. Its edges are never raised, and its consistence is inconsiderable, so that it is easily torn, broken down, and reduced to a state of diffuence: in this state it becomes grumous, and discolours the serum. It is also remarkable for the absence of all buff, which is rarely met with in typhus, in measles, in scarlatina, or in small-pox, unless there has been some inflammatory complication; and even when it does exist, as in confluent small-pox, with large collections of pus, the buff is soft and gelatinous, and, by expression of the serum, is easily reduced to a thin pellicle. This defect of fibrine he conceives to be the cause of the great tendency to hemorrhage, and to that stasis or congestion so remarkable in typhus fever, scarlatina, and other diseases dependent on morbid poisons.

The facts and arguments which have been adduced prove that morbid poisons act in all instances not capriciously, but according to certain definite and specific laws, modified by the influence of climate, temperament, or the magnitude of the dose: also, that they mingle with the blood, with which they continue in latent combination a certain but varying period of time; and likewise that many of them are capable of coexisting together in the same system. A knowledge of these facts is necessary to the proper understanding of this class of diseases, and it is hoped that by their application many of the difficulties which have hitherto obscured the doctrines of fever, of syphilis, of hydrophobia, and of many other diseases incident to this class of morbid poisons, may be removed, and that this portion of medical science may be placed on a surer foundation, if not on a permanent basis.

Deaths from Zymotic Diseases.—The average annual rate per cent. of mortality in Great Britain for the past seventeen years is represented by 2.245; *i. e.*, nearly 22 per 1000, or 1 in 45 of the population. This statement is given as a fact by which the student may compare the numerical statements which are made in estimating the fatal nature of individual diseases, or of diseases considered in classes.

With regard to diseases of the Zymotic class, it may be stated generally, that from 21 to 26 per cent. of the total number of deaths which take place in Great Britain during a year are due to diseases of this class. Generally speaking, also, they may be arranged in the order of their greatest fatality, as follows, namely: (1.) Cholera, typhus, and other forms of continued fever; (2.) Scarlatina, whooping-cough, measles, croup, small-pox, dysentery, erysipelas; (3.) The other diseases of this class are less fatal; and it has been observed that of late years small-pox, influenza, and typhus fever are less fatal than they used to be.

Under the class of Zymotic diseases the following orders are to be distinguished and described, namely:

ORDER 1. MIASMATIC DISEASES—*Miasmatici*.

ORDER 2. ENTHETIC DISEASES—*Enthetici*.

ORDER 3. DIETIC DISEASES—*Dietici*.

ORDER 4. PARASITIC DISEASES—*Parasitici*.

CHAPTER II.

PATHOLOGY OF THE Miasmatic Order of Zymotic Diseases.

THE diseases to be described under this order acknowledge at least three sources or modes of origin; while they are all mainly propagated, disseminated, communicated, or diffused through the agency of contaminated persons, food, water, or other agents, or through infected air. The poisons, miasms, gases, germs, active principles, or morbid agents may be arranged under three classes, namely,—1st. Paludal malarious poison; 2d. Animal malaria poison; 3d. Specific disease poisons. The diseases they engender are attended by a febrile state, which may assume various forms or types.

Paludal Malarious Poison.—This poison arises from marshy land in particular conditions, such as decomposition, under the influence of partial moisture, and of heat above 60° Fahr. If the land is perfectly dry or perfectly flooded the poison is not generated. It is a material poison. It may be wafted along with the wind, and so induce fever at a distance from the place where the poison is generated. It may also be intercepted by a belt of trees. It appears to be most intense near the surface of the ground. The diseases usually attributed to this endemic source, and which were formerly so destructive, have almost disappeared from this country. The reason of this may fairly be ascribed to the improved drainage both of the towns and of the agricultural districts. The fact may be proved, did space permit; and the practical inference leads one to hope for still more immunity from diseases arising from this source, if the “proper authorities” direct further efforts in this direction. “Within the last half century land-draining and town-sewering have ripened into sciences. From rude beginnings, insignificant in extent, and often injurious in their effects in the first instance, they have become of the first importance. Land has, in many instances, doubled in value; and town-sewering, with other social regulations, have not unfrequently prolonged human life from five to fifty per cent., as compared with previous rates in the same district.” “Agues (and malarious cachexiæ) are reduced. Since 1840 an annual mortality in English towns of 44 in 1000 has been reduced to 27; an annual mortality of 30 has been reduced to 20, and even as low as 15; and human life has now more value in England than in any other country in the world—a result entirely due to better sanitary arrangements” (Rawlinson, *Journal of Society of Arts*, March 21, 1862, vol. x, p. 276).

The time, indeed, appears to have arrived when accurate sanitary statistics should not only be kept for all branches of the public service, but also by all corporations, municipalities, boards of commissioners, and parish vestries, for the population within their respective jurisdictions. Such statistics should be published at least once every year, as the natural history of the population (*Sanitary Statistics, B., International Statistical Congress. London, 1860. Second Section.*)

Animal Malaria Poisons or Effluvia.—Animal effluvia arise from the decomposition of the exhalations, excrements, or excretions of individuals (whether of mankind or of the brute creation), of filthy habits, or when crowded in confined spaces. Such poisons appear to be more limited in the causation of disease than the paludal poisons just noticed. They are developed in situations where numbers are crowded together, as in prisons, hospitals, besieged towns, camps, ships, and such-like places. Winter is known to be favorable to their development and deleterious influences. They are sedative or depressing in their actions, and while they lower the energies of the nervous system, they tend to corrupt or poison the blood, surcharging it with decomposing organic compounds.

Ephemeral febrile states are produced, and such conditions of ill-health are thereby established, so that a certain proclivity to the specific communicable diseases seems to be entailed. Conditions of the constitution are brought about by which a certain receptivity or disposition of the blood is produced, rendering it more liable to undergo those changes which it undoubtedly undergoes during the progress of the specific diseases about to be noticed. Thus we have a predisposition to certain diseases brought about, and especially to such specific febrile affections as *typhus fever, dysentery, cholera*, and the like. Dr. Carpenter (than whom we have not a better physiological authority), in an interesting paper on the "Predisposing Causes of Epidemics," shows that the conditions which tend to bring about the specific miasmatic diseases of the Zymotic class are referable to three categories: (1.) Conditions which may introduce into the system decomposing matter that has been generated in some external source. (2.) Conditions which may occasion an increased production of decomposing matter in the system itself. (3.) Conditions which obstruct the elimination of the decomposing matter normally or excessively generated within the system, or abnormally introduced into it from without.

The decomposing matters generated in external sources may be enumerated as putrescent food, water contaminated by sewerage or other decomposing organic substances, and air charged with miasmatic emanations. The constant breathing of such putrescent effluvia may, by communicating a putrescent tendency to the blood, render it more prone to the changes by which specific poisons are multiplied. Ill-health and consequent receptivity for any specific disease poison are developed by the degeneration of the tissues within the body, such as occur in the puerperal state, after severe injuries, and as a consequence of excessive muscular exertion; and also by an insufficient supply of air, a high external temperature,

and the ingestion of alcoholic drinks. Each and all of these causes tend to induce morbid conditions of the blood, a tendency to putrescence, and a condition of ill-health of the body.

Specific Disease-Poisons.—The matter by which the specific miasmatic diseases are communicated and propagated is solely derived from the body of the similarly diseased human or animal being; for there, during the course of the specific disease, is the soil in which the specific poison is bred, to multiply and propagate its kind. It is not yet clearly established how far the bodies of animals may not be a soil for the propagation of diseases communicable to man. (See the Sections on "Small-pox," and "Cow-pox.")

The diseases of the lower animals are not sufficiently studied by us. The diseases of plants are almost entirely neglected. Yet it is clear that until all these have been studied, and some steps taken to generalize them, every conclusion in pathology regarding the nature of the propagation and dissemination of specific miasmatic, and even of parasitic, dietic, and enthetic diseases, must be the result of a limited experience from a limited field of observation. How do we know that the blights of plants, or the cause of them, are not communicable to animals and to man? We know how intimately related the diseases of man and animals are with famines and unwholesome food; and of famines with the diseases of vegetable and animal life, as much as with the destruction and loss of food.

Dr. Carter, of Bombay, has shown that there is in India a very singular, and although strictly endemic disease, yet a very prevalent one, which occurs in the hands and feet, especially the latter, and which it is probable is really of the nature of a "blight," in so far as it is owing to the implantation in the tissues of "sporules or germs," which in the progress of development commit irremediable ravages on the affected parts, leading ultimately to entire disorganization of the tissues. It is known as the "fungus disease of India," originally described by Dr. Carter in the *Transactions of the Medical and Physical Society of Bombay*, No. 6, new series for 1860. (See the account of "Parasitic Diseases" at the end of this volume.)

On the relations between the diseases of man and animals, and especially in connection with food, the reader is referred to a series of papers by the author, in the *Medical Times and Gazette* for 1857.

Dr. William Budd, of Bristol, has also recently directed attention to the occurrence of malignant pustule in *England*, in a paper read at the great meeting of the British Medical Association in London, in August, 1862. He has shown that the disease has not been so uncommon in England as had been supposed—that it is common and very fatal to oxen and sheep in this country—that in man and in sheep the disease is identical—that it is communicable to man by direct inoculation, and also by eating the flesh of the animals affected—that it may be conveyed and disseminated by the bites of insects, such as gnats—and that the disease may be re-communicated from man to animals (*Brit. Med. Journal*, January 24, 1863).

There are some peculiar and characteristic features especially

pertaining to the specific diseases of the Miasmatic order which require special notice as introductory to a description of the individual diseases: First, *They suddenly spring up in a locality—under unfavorable sanitary conditions.* Second, *They may rapidly spread at irregular intervals, so as to incapacitate or destroy great numbers of people.* These two marked and striking features are technically described as being due respectively to *Endemic* and *Epidemic influences*, the nature of which will be considered in the following chapter.*

CHAPTER III.

ON THE NATURE OF ENDEMIC, EPIDEMIC, AND PANDEMIC INFLUENCES.

Endemic Influences result from those conditions or agencies peculiar to a locality which favor the development of various miasmatic diseases, and may thus account for their sudden origin. Such diseases are then said to be *endemic*. These endemic influences, for the most part, are exerted by the geological properties of a district, and are traceable to the constitution and state of the soil, water, and air; to elevation above the level of the sea, vicinity of sea, rivers, or stagnant water, woods, and vegetation; variations of temperature, prevalent winds; in connection with avocations, modes of life, quality of food and quantity, as modified by moral agencies, such as indolence or activity; privation and comforts, filth or cleanliness of people; together with their habits of life and employments, ignorance or mental culture; and lastly, their social, moral, religious, and political conditions. It may be shortly stated in illustration, that endemic influences become mainly active through the following conditions, namely: 1st. That the specific poisons by which the communicable diseases, such as small-pox or typhoid fever, propagate their kind are never totally in abeyance. 2d. The specific communicable diseases are constantly extant somewhere, and only under conditions favorable to their dissemination do they spread or become epidemic. Although their germs, specific gases, active principles, or media of propagation and development may lie dormant or latent for a time, it is not to be inferred that they have ceased to exist. 3d. The history of all the specific communicable diseases demonstrates the same alternations of slumber and activity; of wide-spread prevalence in one place, while neighboring places may remain free; and finally, the same successive invasion of neighboring places, such that the pre-

* Deputy Inspector-General Dr. Lawson has recently called the attention of the profession to what he considers to be *oscillations* of influences over the whole world, determining febrile diseases. To such influences he gives the name of "Pandemic." (*Transactions of Epidemiological Society* for 1862; also, *Statistical, Sanitary, and Medical Reports of the Army Medical Department* for 1861.)

vailing disease only begins to prevail in the new locality after it has already died out in the old. 4th. One element remains constant in the history of endemic influence, and that is *the specific morbid poison* which is the origin of each case. It is susceptible of transmission from place to place, gathering strength as it proceeds, again to die out or become dormant, so that its track is with difficulty followed or traced out. 5th. In large cities such specific poisons are always more or less active, and their diseases always present; but in the country districts they only now and then occur. The occurrence of long intervals of rural exemption is not traceable to any feebleness of the poison to act; for when the disease does become developed in these places, the ratio of persons or of animals attacked is incomparably greater than is ever seen in cities under like circumstances (see Professor Acland's account of the fever in Great Horwood, in 1857-58; and Dr. William Budd, of Clifton, regarding fever at North Tawton; and his most instructive little book *On the Propagation of Typhoid Fever*). 6th. In large towns the sewers are constantly charged with the *materies morbi* of specific diseases always abounding in towns. In small villages, and other places where no sewers exist, the air only may be infected, or the water contaminated, by the direct or indirect importation of cases of specific disease or their equivalents—the poison itself—so that the organic impurities, the dung-heaps, the open soil which surrounds the dwellings of the patients, the cess-pools, and the privies common to several houses, gradually but eventually become impregnated with the specific poison of the disease. Thus the atmosphere of the village may become incomparably more virulent than the atmosphere of the sick-chamber itself. Hence the rapid epidemic spread of the miasmatic diseases in the limited space of rural villages; and which gives rise to the popular error, that such diseases are invariably contagious in country places, and only rarely so, or by exception, in cities or large towns. 7th. All these specific diseases multiply their kind after similar modes of propagation. 8th. All of them establish a constant series of morbid changes and lesions, and always issue in the reproduction of its own specific germ, miasm, gas, morbid poison, or active principle by which it propagates its kind. Thus small-pox propagates small-pox; measles multiplies measles; scarlatina reproduces scarlatina; typhoid fever breeds typhoid fever; typhus, typhus; and so on. In the terse language of Dr. William Budd,—“What small-pox and measles were in the Arab in the days of Rhazes, they still are in the London Cockney of our own time. What they are in the London Cockney, they are in the wild Indian of the North American prairie, and in the Negro of the Gold Coast. To all the other specific communicable diseases, as far as our records go, the same remark applies. In races the most diverse, under climates the most various, age after age, through endless generations of man, these diseases pass down through the human body (sometimes through animals—*e. g.*, ovine small-pox?), perpetuating their own kind, and each maintaining its separate identity by marks as specific as those which distinguish the asp from the adder or the hemlock from the poppy.” Such being the case, it is difficult to conceive (as Drs. Watson and William Budd

most justly observe), "*that diseases of whose propagation this is the history can ever be generated in any other way.*" Most of these miasmatic diseases also are peculiar to man; while animals on their part are infested by a whole brood of communicable diseases, no less specific in their kind, each distinct from the other, and most of them, although some may be communicable to man, are incapable of multiplying in the human body. Cattle appear to be subject to a variety of malignant and communicable fevers from which man is altogether exempt. 9th. Certain receptive conditions, or a predisposition (the nature of which is unknown), exists in individuals, which appear essential to the development of the specific poisons and the establishment of the disease; and immunity against a repetition of the disease is generally conferred by one attack of the same disease—an immunity which has been proved by experiment on an enormous scale with regard to small-pox; and with regard to the other diseases of this kind, the belief in such immunity is deduced from extensive observation. But the immunity acquired by one attack of any of these diseases is of no avail against the rest. Measles, for example, renders the body proof against measles, but leaves it as open to small-pox as before, and so on of the rest. 10th. With regard to fermentation, putrescency, or decomposition, there is some reason to believe (as shown in the previous chapter) that it may quicken the activity or facilitate the development of specific morbid poison, in the way of a predisposing cause. Dr. Budd, however, believes that this effect has been much overrated, notwithstanding the observations of Dr. Carpenter referred to in the preceding pages. Nevertheless, there is no small amount of circumstantial evidence tending to show that endemic conditions may be thus far favorable to the propagation of specific diseases, even to the extent of epidemics, in consequence of the predisposing agency of putrescent emanations; and on the other hand, both endemic and epidemic influences are often held in abeyance by the tendency to decay, decomposition, and destruction of the specific germs, miasms, gases, or disease-poisons themselves. They are stamped with the tendency to change and to perish. Like all organic substances which propagate from minute or invisible beginnings, myriads perish for one that is fruitful. This is especially demonstrable in respect of the Parasitic diseases, whose germs would overrun the world if they all came to maturity: but the extinction or the dispersion of the specific poisons is abundantly provided for through the operation of many natural causes; and by imitating some of these operations of Nature we may be able eventually to exterminate, or, at all events, greatly to modify the severity and reduce the mortality from many of these diseases.

The belief in the spontaneous endemic origin of the specific miasmatic diseases rests on evidence entirely *negative*—namely, the fact that cases do spring up in which it is impossible to trace the disease back to a personal source of specific propagation and dissemination—an event which is inherent in the very nature of these diseases. For the active principle of the poison is invisible, although the matter that is known to contain it may be capable of isolation and inoculation, as in small-pox; yet the existence of the specific disease-poison

is known to us by inference only. Again, we know that ample provision is made and ways are open for the dissemination of the active agent of propagation in a thousand unseen modes, so that it is obvious that the precise sort of infection and its track must often baffle the wisdom of man to discover or trace out.

Cases thus constantly arise which appear to give countenance to the belief that the disease has had a spontaneous origin—*sporadic*, as it is termed. Numerous cases of small-pox occur which can never be traced to their source, or to communication with persons similarly diseased; yet the history of small-pox is decisive against the notion of its spontaneous origin; and if of small-pox, so for all the other specific Zymotic diseases of the same nature. Dr. Watson has well observed that "the small-pox never occurs except from contagion. *It was quite unknown in Europe till the beginning of the eighth century.* No mention of any such malady is to be found in the Greek or Roman authors of antiquity. Now, whatever may have been the deficiencies of the ancient physicians, they were excellent observers and capital describers of disease; and it is impossible that a disease so diffusive, and marked by characters so definite and conspicuous, should have escaped their notice, or have been obscurely portrayed (if known) in their writings. On the other hand, Mr. Moore, in his learned and interesting *History of Small-Pox*, has shown that it prevailed in China and Hindostan from a very early period—even more than a thousand years before the time of our Saviour. That it did not sooner extend westward into Persia, and thence into Greece, may be attributed partly to the horror which the disease everywhere inspired, and the attempts that were subsequently made to check its progress, by prohibiting all communication with the sick, partly to the limited intercourse which then took place among the Eastern nations, but principally to the peculiar situation of the regions through which the infection was diffused, separated as they are from the rest of the world by immense deserts and by the ocean" (Watson, *Lectures on the Practice of Physic*, 3d edition, vol. ii, p. 709). "If anything were wanting," writes Dr. Budd, "to show what is the true inference to be drawn from these events, it would be found in the fact that, *once imported into the West*, it spread with the most fearful rapidity and havoc; and that while almost all men are prone to take the disorder, large portions of the world have remained for centuries exempt from it, until at length it was imported, and that then it infallibly diffused and established itself in those parts. In this country the (endemic) conditions for the spread of the disease existed in the most intense degree, as was shown by the event when the disease was once introduced. The long lapse of ages during which we remained entirely free from small-pox showed, with equal clearness, that, until this introduction occurred, all the conditions favorable to the development of small-pox were powerless to cause a single case. The spectacle witnessed in Europe was repeated over again in the Western World in a still more striking way. Our knowledge of the events here is precise and sure. There was no small-pox in the New World before its discovery by Columbus, in 1492. In 1517 the disease was imported into St. Domingo. Three

years later, in one of the Spanish expeditions from Cuba to Mexico, a Negro covered with the pustules of small-pox was landed on the Mexican coast. *From him* the disease spread with such desolation that within a very short time (according to Robertson) three millions and a half of people were destroyed by it in that kingdom alone." "Again, small-pox was introduced into Iceland in 1707, when sixteen thousand persons were carried off by its ravages—more than a fourth part of the whole population of the island. It reached Greenland still later, appearing there for the first time in 1733, and spreading so fatally as almost to depopulate the island," (Budd, l. c., p. 35, *et seq.*) No common conditions of human life gave rise to such phenomena. Propagation from the actual poison of a pre-existing case was the one necessary and all-sufficient condition for these endemic outbreaks and their epidemic prevalence. The precise mode in which the miasmatic diseases, with their specific poisons, first came into existence is beyond our *ken*—hidden from us as yet by a veil, and remaining an inscrutable, at least an unpenetrated, mystery. But everything tends to show that once created, they all propagate only in one way—namely, by continuous succession.

Defective ventilation, inasmuch as it is always injurious to health, always aggravates disease, and so promotes the endemic influence. With regard to any influence it may have on the development and spread of communicable disease, it may be noticed that it does not equally help *all* communicable diseases to develop themselves and to spread. Commonly it seems to operate in proportion as the specific miasmatic disease is one which imparts specific poison-properties to the general exhalations of the sick. The significance of defective ventilation is not likely, therefore, to be quite the same where typhoid fever, cholera, or dysentery are the prevailing diseases, as where the disease is typhus fever, scarlatina, small-pox, or diphtheria. In the cases of typhoid fever, cholera, and dysentery, any defect of ventilation would become more and more important in proportion as the bowel discharges of the sick were not promptly removed from within doors, or as, from other causes, there was fecal effluvia or excrements suffered to remain in the dwelling (Simon, *Third Report of the Medical Officer of the Privy Council*, 1860, p. 10).

Epidemic Influence.—The second characteristic feature peculiar to some of the miasmatic order of Zymotic diseases is, that they sometimes spread rapidly, so as to incapacitate and destroy great numbers of the people. The disease is then said to be *epidemic* (*επι*, upon, and *δημος*, the people). No subject has afforded greater scope for speculation than the origin, cause and progress of epidemics. It is vain to speculate upon the subject; and, in the words of Dr. Wood, of Philadelphia, "all we can say with certainty regarding epidemics, is that there must be some distempered condition of the circumstances around us—some secret power that is operating injuriously upon our system—and to this we give the name of *epidemic influence* or *constitution*," and which is believed to predispose towards the receptivity of specific disease-poisons. The observations of Mr. Simon lead to the belief that the prevalence of external con-

ditions, tending in certain localities to determine a specific decomposition of excrement, communicable to other organic substances and infecting the air, is an essential element in an epidemic period.

The most recent speculation regards the discovery of a peculiar atmospheric condition, ascribed to a principle called *ozone* or *osmazone* (ὄσμις, stink, or ὀσμή, smell), of which, as yet, we know nothing definite, although many subtle instruments and apparatus are in use to detect and measure the amount of this principle in the air.

A careful study of the effects of the *epidemic influence* appears to warrant the enunciation of certain laws which seem to regulate its operations. These laws may thus be condensed :

Laws of Epidemic Influence.—(1.) This influence frequently predisposes to diseases, apparently independently of any other known cause, as in the case of influenza and cholera. It makes itself manifest by appearing to give increased energy to causes which produce particular diseases ; so that small-pox, scarlatina, typhus, and the like, sometimes rage with great violence as epidemics. It also appears to predispose to new and anomalous forms of disease, as witnessed in the furunculoid epidemic which recently prevailed both in Europe and America, from 1849 till 1852. (2.) Sometimes the *epidemic influence* manifests itself by a certain type or direction which existing diseases appear to take. Thus, at one period diseases take a low, or what is called a *typhoid* type, so that depletion is not tolerated ; at another time an *inflammatory* tendency predominates, and antiphlogistic treatment is required. At one period there is a tendency in disease to complicate its course by a disposition to affect particular organs. At one time head affections predominate ; at another time affections of the chest, or of the alimentary canal, complicate the course of a prevailing disease. Consequently the same disease may demand very different, and even opposite, modes of *management*. (3.) During epidemics other diseases are apt to assume more or less of the prevailing epidemic features. Thus, when cholera prevails, looseness of the bowels often complicates the course of other affections. When influenza prevails, catarrhal complications increase the danger of other diseases. Ill-health of any kind, therefore, favors the action of the epidemic influence. (4.) Some change in the character of prevailing diseases of a constant and recurring kind often indicates the approach of an epidemic and the prevalence of the epidemic influence. (5.) The first effects of the *epidemic influence* are usually the most violent and marked, and the cases of the epidemic disease become mild as the *epidemic influence* passes away. (6.) The *epidemic influence* sometimes disappears entirely after a short prevalence ; sometimes continues, with irregular intermissions, for two, three, four, or even six years, or longer. Influenza and cholera are examples. (7.) An epidemic tendency, after continuing for several years, may give place to one of a different kind, which, in its turn, may again give place to the first. *Malarious fevers, yellow fever, and typhus*, illustrate this in America. The eruptive affections seem to run in somewhat similar cycles. After the introduction of vaccination the *small-pox* seemed for many years to be almost entirely subdued ; but more recently again the disease has seldom been en-

tirely absent from among us, alternating as an epidemic now and then with *measles*, *scarlet fever*, and *typhus*. We look forward to the time when vaccination, enforced by law, will completely eradicate the disease. (8.) The lower animals are also subject to epidemic influences; and seasons of unusual fatality among them have coincided with those in which the human race have suffered. This fact has been shown in an elaborate and erudite analysis of the census of Ireland, by Sir William R. Wilde, of Dublin, the diseases of the population having been recorded at the time.

Pandemic Influences.—The expressions of the hitherto prevailing doctrines regarding *endemic* and *epidemic* influences appear so unsatisfactory to many minds, and leave many circumstances regarding the spread of diseases unexplained, that attention is being directed to more comprehensive views and investigations of the questions involved in the preceding paragraphs. An ingenious theory has been propounded by Deputy Inspector General Dr. Lawson, who has attempted to establish the occurrence, between 1817 and 1836, of a series of *oscillations of febrile diseases*, following each other over the world with amazing regularity. The mode of occurrence of such febrile diseases he attributes to a cause or influence which, from its extent and *progressive* character, he names a "*pandemic wave*," to distinguish the influence from that usually understood as *epidemic*, referring to a single form of disease affecting a limited space. Under the influence of this *pandemic wave* Dr. Lawson believes that there is a constantly progressive tendency to the development of all *endemic* febrile diseases in the Atlantic and Western parts of the Indian Ocean, from south or southeast to north or northeast.

But the facts and data on which this theory is made to rest are not of sufficient number, and many of them are not sufficiently trustworthy, to rest a judgment upon. In not a few instances a totally different interpretation may be given to that which Dr. Lawson has assigned to them. Although, therefore, it may be premature to propound such a theory, especially as it is still open to the verdict of "not proven," yet the expression of it is calculated to do good by drawing attention to the subject and to the comprehensive, world-wide range which must be given to such investigations; and to whom can Science look with more hope for results than to the medical officers of Her Majesty's British and Indian armies?

A successful study of these peculiar and characteristic features of miasmatic diseases—namely, the *endemic*, *epidemic*, and *pandemic influences*—is of the utmost importance to the student. He will learn to appreciate how much and successfully mortality may be diminished by well-directed hygienic measures, such as cultivation and improvement of the soil, extension of commerce, improvements in diet and the social circumstances of the lower classes—especially in regard to cleanliness, ventilation, and domestic management of improved dwellings, and efficient sewerage; care in the separation and treatment of the sick when in numbers; and the use of strict measures of a prophylactic kind suited to the circumstances of the

case. Next to large towns, the health of the Army is of the greatest importance, especially when we consider the tendency that exists to a high rate of mortality in that service. In the military age (which is the age between eighteen and forty) the mortality of the general population in England is less than one per cent. per annum. The mortality of the British army is much above this. On Home service it has had a mortality double that of the civil population at the corresponding ages; and seven-ninths of the entire mortality among the infantry of the line has arisen from diseases of the Zymotic class. Disease and mortality are much greater during campaigns, when more than twenty-two per cent. are constantly on the sick list. The causes of high rates of mortality require constant investigation, by carefully observing, recording, and comparing the facts over a sufficiently large area; thus arriving at certainty as to the causes, and whether they can be mitigated or removed.

An observation of great interest in connection with *animal malaria poison*, as well as with *epidemic influences*, may be appropriately referred to here. It seems clearly proven, especially by the valuable and decisive observations of Dr. William Budd, of Bristol, that the communicable poisons of typhoid fever and of cholera are capable of being imported or carried from place to place by persons who have the disease. Dr. Budd's history of the North Tawton fever and its offshoots (*Lancet*, July 9, 1860) is most conclusive on this point. His arguments are also cogent to the general effect that *specially* the bowel discharges are *means* by which a patient, whether migrating or stationary, can be instrumental in disseminating typhoid fever and cholera. Mr. Simon makes the important remark, however, that these bowel discharges may not be the *sole means* of multiplying and disseminating these diseases; although, provisionally, the conclusions of Dr. Budd must be acted upon in their present unqualified form; while it is of the greatest practical importance to learn, as exactly as possible, whether it is in all states of the disease, and under all circumstances, that the bowel discharges of typhoid fever and cholera can communicate and multiply the means of dissemination. In illustration of such possible contingent results, Mr. Simon refers to some interesting and important experiments made in 1854 by Professor Thiersch, of Erlangen. These experiments seemed to show that cholera evacuations, *in the course of their decomposition*, either acquire the power of communicating or multiplying their specific poison, or that the specific poison inherent in them becomes intensified by decomposition (Zymosis?). That the decomposition or change may begin even in the bowels, after the secretion and accumulation of the material in them, as well as in cesspools, seems to be possible; and perhaps, as Mr. Simon justly remarks, may furnish an explanation of the many cases in which human intercourse has apparently disseminated the disease. For, according to the observations of Professor Pettenkofer at Munich, and Professor Acland at Oxford, it would seem that during cholera periods the immigration of persons suffering apparently only from diarrhoea has been followed by outbreaks of cholera in places previously uninfected; and Professor Pettenkofer ascribes this fact to an

influence (Zymotic?) exerted by the decomposing feces of such diseased persons, in the cesspools and adjoining soil of ill-conditioned places to which they go. Specific poison properties of this kind would thus arise, and probably extend to the pollution of well-waters of such soils, and might render them, if swallowed, capable of exciting cholera, or typhoid fever, or dysentery, by direct *contagion*; and so any of these diseases would thus have all the appearance of having arisen *de novo*. It is encouraging to sanitary reformers, as Mr. Simon justly remarks, that cases of the apparent introduction of cholera contagion by human intercourse are essentially different from cases of the dissemination of such specific diseases as small-pox or measles. The multiplication of the specific poison in the latter diseases takes place exclusively within the human body. The multiplication and dissemination of them have no immediate dependence on differences of medium; but wherever human beings can cross one another's path, to the susceptible or unprotected person these specific diseases may be communicated. On the other hand, it seems really to be the fact that the cholera poison (and probably, also, typhoid fever poison and dysentery), if it can at all be multiplied within the body, almost certainly has its great centres of multiplication *elsewhere*; namely, in those avoidable foci of corruption where excrement accumulates and decays. Military authorities ought to remember this fact. They have had abundant evidence of it in the old camping-grounds of the Indian army, as well as when following the route and encamping on the ground previously occupied by retreating armies. For disseminating the disease and multiplying the poison, foulness of medium seems indispensable; and it is no ordinary foulness which will impart to air, food, or water the Zymotic action of decomposing excrement. The common taint is something specific. Therefore, as regards cholera, it seems highly probable that the immigration of infected persons might occur to a very great extent without exciting epidemic outbreaks, if such immigration were only made into places of irreproachable sanitary conditions, especially as regards water supply and the continuous removal of house refuse or camp filth. (Compare Simon in his Public Health Reports—especially *second* and *third*—relative to the people of England; also Pettenkofer, Acland, and Thiersch, as quoted by Simon, p. 3 of his *third* Report, 1860.)

CHAPTER IV.

MANAGEMENT OF EPIDEMICS; AND ON PROCEEDINGS WHICH ARE ADVISABLE TO BE TAKEN IN PLACES ATTACKED OR THREATENED BY EPIDEMIC DISEASES.

THE practical questions immediately involved in the exposition which has been given of the nature of Zymotic diseases in general, and of the miasmatic order of these diseases in particular, is

contained in the following statement, namely: That it is possible to extinguish the greater number of epidemic diseases, however intense or abundant may be the atmospheric or other agencies which constitute their potential causes, by remembering,—(1.) That the living body of the diseased persons is the soil on which the communicable disease breeds the poison by which the specific disease is multiplied and propagated: (2.) That excretions from an infected person, especially such excretions as are immediately related to or flow from parts affected with specific lesions, probably contain the most active elements of the specific poison by which the disease may be disseminated: (3.) That such active elements, germs, poisons, miasms, gases, or noxious agents may contaminate the drinking-waters of a district, or may infect the atmosphere, or lie dormant for variable and unknown periods of time, just as seeds dry up and preserve their vital properties: (4.) To follow out zealously the hygienic measures which flow from these statements, and so prevent the propagation of specific diseases: (5.) To preserve as much as possible the blood of every individual in that state which shall prevent these poisons from finding the conditions of their development within the body: (6.) That these ends are to be attained on the one hand by preventing the production of fermentable matter in or out of the body; and on the other hand by promoting its removal and chemical destruction or decomposition, when it is inevitably generated, and by a free supply of pure air, and by the reduction of that air to the lowest temperature at which the condition of the individuals will allow it to be safely inhaled. Preventive measures based upon these principles are of the utmost importance, so much so that the most eminent members of the medical profession in London and elsewhere concur in the views and opinions of Dr. William Budd, unanimously cherishing the maxim, that, “except under the pressure of great military straits, no army ought ever to suffer on a large scale from this great group of communicable diseases, and especially such as are disseminated by intestinal discharges.”

The following detail of proceedings advisable to be taken in places attacked or threatened by epidemic diseases are given mainly from a memorandum drawn up by John Simon, Esq., the Medical Officer of the Privy Council, and published in his *Third Report on the Public Health in England in 1860*:

1. Wherever there is prevalence or threatening of cholera, diphtheria, typhus, or any other epidemic disease, it is of more than common importance that the powers conferred by the Nuisances Removal Acts, and by various other laws for the protection of the Public Health, be vigorously, but at the same judiciously exercised by those in whom they are vested; and with regard to armies, that the instructions relative to the guidance of the Medical Officer in sanitary matters, contained in the *Army Regulations*, be duly carried out, on the principle that the executive should act under authority, in order to carry out the required measures efficiently.

2. If the danger be considerable, it will be expedient that the local authorities in civil life, and the commanding officers of armies, brigades, divisions, and regiments, in military life, avail themselves,

as soon as possible, of the medical advice within their reach, in taking measures of prevention and protection against the spread of epidemic influences.

3. Measures of precaution for prevention and protection are equally proper for all classes of society, civil and military. But it is chiefly with regard to the poorer civil population—therefore chiefly in the courts and alleys of towns, and at the laborers' cottages of country districts—that local authorities are called upon to exercise the utmost vigilance, and to proffer information and advice. Common lodging-houses which are sub-let in several small holdings, always require particular attention.

4. Wherever there is accumulation, stink, or soakage of house refuse, or of other decaying animal or vegetable matter, the nuisance should as promptly as possible be abated, and precaution should be taken not to let it recur. Especially all complaints which refer to *sewers and drains*, or to *foul ditches and ponding of drainage*, or to *neglect of scavenging*, should receive immediate attention. The *trapping of house drains and sinks* and the state of *cesspools and middens*, should be carefully seen to. In *slaughter-houses*, and other places where beasts are kept, strict cleanliness should be enforced.

5. In order to guard against the harm which sometimes arises from disturbing heaps of offensive matter, it is often necessary to combine the use of chemical disinfectants with such means as are taken for the removal of filth; and in cases where removal is for the time impossible or inexpedient, the filth should always be disinfected. Disinfection is likewise desirable for unpaved earth close to dwellings, if it be sodden with slops and filth. Generally, where cholera or typhoid fever is in a house or barrack, hospital or hut, the privies especially require to be disinfected.

6. Sources of water-supply should be carefully and efficiently examined. Those of them which are in any way tainted by animal or vegetable refuse—above all, those into which there is any leakage or filtration from sewers, drains, *cesspools*, or *foul ditches*—ought no longer to be drunk from. Where the disease is cholera, diarrhœa, or typhoid fever, it is especially essential that no foul water be drunk.

7. The washing and lime-whiting of uncleanly premises (houses, huts, hospitals, barrack guard-rooms, and the like), especially of such as are densely or multifariously occupied, should be pressed with all practicable despatch.

8. Overcrowding should be prevented. Especially where disease has begun, the sick-room should, as far as possible, be free from persons who are not of use or comfort to the patient.

9. Ample ventilation should be enforced. Window frames should be seen to, (1.) That they may be made to open, if not so made; and (2.) That they be kept sufficiently open. Especially where any kind of specific disease, communicable by infection of the air, has begun, it is essential, both for patients and for persons who are about them, that the sick-room and the sick-house or hospital be constantly and efficiently traversed by streams of fresh air. This is especially necessary at night, and steps should be taken to insure

efficient ventilation even at some real or imaginary expense of comfort.

10. The cleanest domestic habits should be enjoined. Refuse matters should never be suffered to remain or to linger within the dwelling, hospital, barrack-room, or hut. Such refuse must *at once* be removed, and at once disposed of, or cast into the receptacle provided for it. All things or utensils which have to be disinfected or cleansed should always be disinfected or cleansed *without delay*.

11. With regard to material substances discharged or separated from the bodies of the sick, special precautions of cleanliness and disinfection are necessary. Among discharges or substances separated from the body which it is proper to treat as capable of communicating disease, are those which come, in cases of small-pox, from the affected skin; in cases of cholera and typhoid fever, from the intestinal canal; in cases of diphtheria and scarlatina maligna, from the nose and throat, and the exhalations from the skin and the lungs saturating clothes; likewise, in cases of eruptive fevers, measles, scarlatina, r  theln, typhus, and the like, the general exhalations of the sick, and especially so of the convalescing, probably in connection with the desquamation of the skin. The caution which is necessary with regard to such matters must of course extend to whatever may be imbued with them; so that bedding, clothing, towels, and other articles which have been in use by the sick, do not become sources of mischief, either in the house to which they belong, or in houses to which they are conveyed. Moreover, in typhoid fever and cholera, the evacuations should be regarded as capable of communicating a similarly specific and infectious property to any night-soil with which they may be mingled in privies, drains, or cesspools (THIERSCH). This danger of multiplying the sources of communicating disease must be guarded against by the chemical destruction, decomposition, or disinfection of all the intestinal evacuations as soon as they are passed from the bowels, and certainly before they are thrown away, and so "let loose upon the world." Above all, they must never be cast where they can run or soak into sources of drinking-water.

12. All reasonable care should be taken not to disseminate disease by the unnecessary association of persons suffering from the specific communicable diseases, either with healthy persons, or in wards of hospitals where patients suffering with other diseases are being treated. This care is requisite, not only with regard to the sick-house, ward, hospital, or ship, but likewise with regard to day-schools, places of public resort, courts of justice, and other places where members of many different households are accustomed to meet.

13. Where dangerous conditions of residence cannot be promptly remedied, it will be best that the inmates, while unattacked by disease, remove to some safer lodging. If disease begins in houses where the sick person cannot be rightly circumstanced and tended, medical advice ought to decide on the propriety or fitness of removing him to an infirmary or hospital. In extreme cases, special in-

firmaries may become necessary for the sick, or special houses of refuge for the endangered.

14. The questions of quarantine ought to be decided by the circumstances of the special case, the preceding principles being kept in view.

15. Privation, as predisposing to disease, may require special measures of relief.

16. In certain cases special medical arrangements are necessary. For instance, as cholera in this country almost always begins somewhat gradually, in the comparatively tractable form of what is called "premonitory diarrhœa," it is essential that, where cholera is epidemic, arrangements should be made for affording medical relief without delay to persons attacked even slightly with looseness of the bowels. So again, where small-pox is the prevailing disease, it is essential that all unvaccinated persons (unless they previously have had small-pox) should very promptly be vaccinated; and re-vaccination should also be offered, both to persons above puberty who have not been vaccinated since childhood, and to younger persons whose marks of vaccination are unsatisfactory.

17. It is always to be desired that the people should, as far as possible, know what real precautions they can take against the disease which threatens them; what vigilance is needful with regard to its early symptoms; and what, if any, special arrangements have been made for giving medical assistance within the district. Especially in the case of small-pox or of cholera, such information ought to be spread abroad by means of printed bills or placards. In any case where danger is great, house to house visitation, or personal inspection of all by discreet and competent persons, may be of the utmost service, both in quieting unreasonable alarm, and in leading or assisting the less educated and the destitute parts of the population to do what is needful for safety.

18. These memoranda relate to occasions of emergency. The measures suggested must be regarded as of an extemporaneous kind. Permanent provisions for securing Public Health have not been in express terms insisted on. In proportion as a district or number of individuals, such as an army or a regiment, are habitually well cared for by its sanitary authorities, the more formidable emergencies of epidemic disease are not likely to arise.

As addenda to these memoranda, the following rules, the observance of which is enjoined by the government of the London Fever Hospital, might well be adopted under similar circumstances in military and civil hospitals:

1. It is of the utmost importance to the sick and their attendants that there be a constant admission of fresh air into the room, and especially about the patient's bed, care being taken to prevent the wind from blowing directly on the patient.

2. Attention to cleanliness is indispensable. The linen of the patient should be often changed, and the dirty clothes, &c., immediately put into fresh cold water, and afterwards well washed. The floor of the room must be cleansed every day with a mop, and all

discharges from the patient immediately removed, and the utensils washed.

3. Nurses and attendants ought to endeavor to avoid the patient's breath and the vapor from the discharges.

4. Visitors must not go near to the sick, nor remain with them longer than is absolutely necessary: they should not swallow their spittle, but clean the mouth and nostrils when they leave the room.

5. No dependence must be placed on vinegar, camphor, or other supposed preventives, which, without attention to cleanliness and admission of fresh air, are not only useless, but by their strong smell render it impossible to perceive when the room is filled with bad air or noxious vapors.

Processes of Disinfection.—These processes have been recommended by Professor Miller, of King's College, London. They cannot supply the place of cleanliness, ventilation, and drainage. They are artificial, and are used for exceptional purposes, the great natural disinfectant being *fresh air*, abundantly and uninterruptedly supplied.

1. For purposes of artificial disinfection, the agents which most commonly prove useful are, chloride of lime, quicklime, Condyl's manganic compounds, and carbolic acid. Metallic salts, especially perchloride of iron, sulphate of iron, and chloride of zinc, are under some circumstances applicable. In certain cases chlorine gas or sulphurous acid gas may advantageously be used; and in certain other cases powdered charcoal or fresh earth.

2. If perchloride of iron or chloride of zinc be used, the common concentrated solution may be diluted with eight or ten times its bulk of water. Sulphate of iron or chloride of lime may be used in the preparation of a pound to a gallon of water, taking care that the water completely dissolves the sulphate of iron or has the chloride of lime thoroughly mixed with it. Condyl's stronger fluid (red) may be diluted with fifty times its bulk of water; his weaker fluid (green) with thirty times its bulk of water. Where the matters requiring to be disinfected are matters having an offensive smell, the disinfectant should be used till the smell has entirely ceased.

3. In the ordinary emptying of privies or cesspools, use may be made of perchloride of iron, of chloride of zinc, or of sulphate of iron. But where disease is present, it is best to use chloride of lime or Condyl's fluid. Where it is desirable to disinfect before throwing away the evacuations from the bowels of persons suffering from certain diseases, the disinfectant should be put into the night-stool or bed-pan when about to be used by the patient.

4. *Heaps of manure* or of other filth, if it be impossible or inexpedient to remove them, should be covered to the depth of two or three inches with a layer of freshly burnt vegetable charcoal in powder. Freshly burnt lime may be used in the same way, but is less effectual than charcoal. If neither charcoal nor lime be at hand, the filth should be covered with a layer, some inches thick, of clean dry earth.

5. *Earth near dwellings*, if it has become offensive or foul by the

soakage of decaying animal or vegetable matter, should be treated on the same plan.

6. *Drains and ditches* are best treated with chloride of lime, or with Condyl's fluid, or with perchloride of iron. A pound of good chloride of lime will generally well suffice to disinfect 1000 gallons of running sewage; but of course the quantity of disinfectant required will depend upon the amount of filth in the fluid to be disinfected.

7. *Linen and washing apparel*, requiring to be disinfected, should, without delay, be set to soak in water containing, *per gallon*, about an ounce either of chloride of lime or of Condyl's red fluid: the latter, as not being corrosive, is preferable. Or the articles in question may be plunged at once into boiling water, and afterwards, when at wash, be actually boiled in the washing water.

8. *Woollens, bedding or clothing*, which cannot be washed, may be disinfected by exposure for two or more hours in chambers constructed for the purpose, and heated to a temperature of 210° to 250° Fahr.

9. For the disinfection of *interiors of houses*, the ceilings and walls should be whitewashed with quicklime. The wood-work should be well cleansed with soap and water, and subsequently washed with a solution of chloride of lime, about two ounces to the gallon.

10. *A room no longer occupied* may be disinfected by sulphurous acid gas, or chlorine gas,—the first by burning in the room an ounce or two of flowers of sulphur in a pipkin; the second by setting in the room a dish containing a quarter of a pound of finely powdered black oxide of manganese, over which is poured half a pint of muriatic acid, previously mixed with a quarter of a pint of water. In either case the doors, chimney, and windows of the room must be kept carefully closed during the process, which lasts for several hours.

CHAPTER V.

DETAILED DESCRIPTION OF THE MIASMATIC ORDER OF ZYMOTIC DISEASES.

SECTION I.—ERUPTIVE FEVERS.—*Exanthemata*.

SMALL-POX.

LATIN, *Variola*; FRENCH, *Variole*; GERMAN, *Blattern*—Syn, *Menzhenpocken*; ITALIAN, *Vajuolo*.

Definition.—*Small-pox in man is the product of a specific and palpable morbid poison, which is reproduced and multiplied during the course of the malady. After a definite period of incubation a remittent fever is established, and followed by an eruption on the skin, and sometimes on the*

mucous surfaces, with other concomitant and occasionally succeeding affections. The eruption on the skin passes through the stages of pimple, vesicle, pustule, scab, and leaves marks or cicatrices on its site. The disease runs a definite course, and as a rule, exhausts the susceptibility of the constitution to another attack.

Pathology.—The theory regarding the development of small-pox is, that a specific poison is absorbed and infects the blood, and after a given period of latency gives rise to "primary fever," which lasts from two to four days, till the eruption appears, when the fever for the most part *remits*. The secondary or specific action of the poison of small-pox makes itself obvious by an eruption on the skin, and also sometimes on the mucous membrane of the eyes, nose, mouth, fauces, and great intestine. The eruption runs a given course—namely, *pimple, vesicle, and pustule*—and when fully out, or at its height, the febrile phenomena, which had remitted, return, and give rise to what is termed the *secondary fever*. The occasionally succeeding morbid conditions are inflammation of the various tissues of the lungs, of the urinary organs, and, lastly, of the areolar tissue of the body generally, which often becomes the seat of an endless number of abscesses.

The occurrence of fever preceding the secondary or specific actions of the poison, or the appearance of the eruption, has scarcely an exception, and, indeed, in some instances it has been of so severe a character as to have destroyed the patient on the first onset. The remission or subsidence of the fever is constant in mild cases, but in the severer forms of the confluent small-pox it sometimes runs on, and is constant. The recurrence of the "secondary fever," and the exacerbation of the fever in severe cases at the time of the maturation of the pock, is also constant. The cause of this secondary attack has long been a difficulty in the pathology of small-pox. Some attribute the fever to the specific nature of the disease, while others consider it to result from the maturation of the pustules, and to be a *suppurative fever—symptomatic*, and dependent upon the local affection.

Another constant phenomenon in the development of small-pox is, that the secondary actions of the poison occasion a peculiar eruption. There are a few rare exceptions, which constitute a variety of small-pox sometimes noticed as the "*variola sine eruptione*." The affection of the mucous membranes is often wanting in mild cases, though rarely absent in severe ones. The poison is also apt to set up many tertiary actions, as inflammation of the lungs, of the urinary organs, of the eye, and of the areolar tissue. Generally it may be mentioned that the state and appearance of the eruption depends in a great measure upon the type and character of the fever, while the type and character of the fever may be modified by the organic functions and condition of the blood, especially as induced by vaccination.

The development of small-pox is traceable through certain stages, namely, 1st. The stage or period of incubation; 2d. The febrile stage, or period of primary fever; 3d. The exudative stage, or

period during which the eruption appears and becomes fully developed; 4th. The suppurative stage, or period of secondary fever.

As the eruption, or formation of the small-pox pustule, is undoubtedly a marked characteristic of the disease, it requires particular description. It has itself certain definite stages in its development. It runs a given course of about eleven days, and in its progress undergoes many mutations. It is at first a pimple, then a vesicle, then a pustule, and lastly it forms the scab or crust. These various changes form so many *stadia* of unequal duration. The first, or stage of pimple, lasts from twenty-four to forty-eight hours; the second, or vesicular stage, four days; the pustular stage, three days; while the last stage, or that of scabbing, lasts three days more, making the whole duration of the *normal* pustule ten or eleven days. There are varieties, however, of this disease, in which the formation of the pustule is irregular, as in the *confluent* and *horn* small-pox. In the latter the two last stages are singularly shortened, or absent altogether.

When the eruption in small-pox is of the "*distinct variety*" (*Syn.*, "discrete"), its first appearance consists of a number of small red pimples, about the size of a pin's head, more or less numerous, but separate and distinct from one another, and scarcely salient. On the second or third day of the eruption the second stage towards the development of pustules commences; and a small vesicle, which gradually enlarges, bound down and depressed in the centre, or *umbilicated*, forms on the apex of each pimple, and contains a clear whey-colored fluid. This vesicular stage lasts about four days, when the vesicle matures or "*ripens*" into a pustule. This process is so gradual that, if you examine the pustule closely about the fifth or sixth day, you may see, at least in many, two colors, viz., a central whitish disc of lymph, set in, or surrounded by, a circle of yellowish puriform matter. "In truth, there is in the centre a *vesicle*, which is distinct from the pus, so that you may puncture the vesicular portion, and empty its contents, without letting out any of the pus; or you may puncture the part containing the pus, and let *that* out without evacuating the contents of the vesicle. The vesicles have even, by careful dissection, been taken out entire" (WATSON). The adherence of the altered cuticle to the cutis at some points, and its separation at others, produces the little compartments or dissepiments spoken of by some writers. These cavities are usually irregular in shape; and all who have examined these multilocular cavities agree in describing the existence of a white substance in them, of the consistence of pulp or thick mucus, and which at first was supposed to be the specific exudation of small-pox. It is now ascertained that it is no pseudo-membrane, but is composed of the deeper and softened layers of the epidermis. This "*disc*" of softened epidermis covers the interior of the pustule, and extends from the centre to the raised circumference of the pustule in diverging rays, forming part of six or eight chambers of nearly equal size. In the structure of this disc the following elements are distinguishable from without inwards,—(1.) Large flat cells; (2.) Large cells not so flat, but more globular, with nuclei; (3.) Nearest

the cutis are the cells and tissue of the *rete mucosum* (GRUBY, GLUGE, RAYER, GUSTAV. SIMON, besides other observers of more early date). Will not some delicate process of organic analysis tell us what the *active principle* of the specific virus of small-pox is,—if it be capable of being so determined? While the maturation of the vesicle into a pustule is going on, a damask red areola forms around each pustule; and as the vesicle fills, the whole face swells, and often to so great a degree that the eyelids are closed. While the maturation is complete, the “*bride*,” which bound down the centre of the vesicle, ruptures, and the pustule now becomes *spheroidal* or *acuminated*. About the eighth day of the eruption a dark spot is seen on the top of each pustule. At that spot the cuticle ruptures, allowing matter to exude, which concretes into a scab or crust; and during this process the pustule shrivels and dries up. The crust is detached between the eleventh and fourteenth days, leaving the cutis beneath of a dark reddish-brown hue—a discoloration which lasts many days, or even weeks. On the face, however, the pustule often penetrates or burrows, so as to cause ulceration of the *rete mucosum*, leaving a permanent cicatrix in the form of a depression or “pit.” The cicatrix thus formed, though at first of a reddish-brown, ultimately becomes of a dead white color.

The small-pox eruption does not appear over the whole body at once, but appears in three successive crops. The first crop covers the face, neck, and upper extremities, the second the trunk, while the third appears on the lower extremities. There is usually an interval of several hours between each crop; and the later the pustules are in appearing on the trunk and lower extremities than on the face and neck, by so much the later they are in maturing, and in disappearing from those parts. When the eruption on the face is declining, that upon the extremities has scarcely yet arrived at its height, so that the hands and feet are then considerably swollen. This is to be regarded as a favorable sign, in so far as it indicates a certain vigor of constitution.

The number of pustules is very various, sometimes not exceeding five or six over the whole body, more commonly from one to three hundred, and occasionally amounting to several thousands. It has been calculated, if ten thousand pustules be counted on the body, that two thousand at least will be found on the face; and accordingly, the number of pustules on the face being in proportion, those on the other parts of the body furnish a fair estimate of the extent of the disease, and of the danger of the patient.

The pustule is subject to many irregularities, both as to its form and course; which gives rise to two very marked varieties of the disease, namely, the *confluent* and the *horn* small-pox. The *confluent small-pox* differs from the *distinct small-pox* in the pimples being small, less prominent, and so numerous that even on the first appearance of the eruption there is hardly any distinct separation between them. The vesicles which form on their apices appear earlier, and their diameters increase more irregularly than in the distinct forms, and often they run one into the other. The pustules, likewise, which are confluent, either remain flat, and do not

rise, or, the areolar tissue rupturing, they form large bullæ or bladders in clusters like a bunch of grapes—a rare variety of the disease (*variola corymbosa*)—and are not encircled with the usual red areola round their base; neither do their fluid contents always acquire the yellow color and thick purulent consistency of the milder disease. Their crusts, moreover, are soft, and do not fall off till many days after the usual period, or not till the eighteenth or twentieth day, or even later. When the desiccation is completed and the crust detached, a deep scar or pit, sometimes an extensive seam, remains, and shows the loss of substance that has taken place, and how destructive has been the process beneath these crusts.

The *horn small-pox* is a variety of the pustule, and is by much the mildest form of the disease. The pustule in this variety passes through the stages of pimples and of vesicle, but on the fifth or sixth day of the eruption, instead of maturing, the pustule shrivels, desiccates, and crusts, and the disease terminates three or more days earlier than in the usual course, and without the occurrence of any secondary fever. This is the form of the disease which so usually follows after vaccination.

Many other varieties have been described by the older authors, which are seldom if ever now seen—for instance, *black small-pox* (SYDENHAM); a *blood small-pox* (MEAD), or *Hemorrhagic*, in which “blood is effused into the vesicles or pustules;” a *sitiquous small-pox* (FRIEND), in which the pustule resembles a small hollow bladder, but contains no fluid. There is one variety, however, which is not uncommon, called the *crystalline* or *pearl pock* (*variola crystallina*), in which the vesicle continues transparent, seldom matures, and has a tendency to become confluent. Every variety of the eruption, when the disease is severe, may be intermixed with petechiæ. Such are the chief features of the disease, so far as the development of the eruption is concerned.

Varieties and Symptoms of Small-Pox.—The species of small-pox to be described are,—(1.) *The Natural or Unmodified Small-Pox*; (2.) *The Inoculated Small-Pox*; (3.) *The Modified Small-Pox*, or *Small-Pox after Vaccination*, or *Varioloid*. Of these in their order; and,

1. *Of the Natural Small-Pox.*

There are several varieties of this species, namely,—1st. The small-pox without eruption (*variola sine eruptione*); 2d. The distinct small-pox (*variola discretæ*); and 3d. The confluent small-pox (*variola confluentes*); 4th. *Semi-confluent*; 5th. *Abortive*; 6th. *Petechial*; 7th. *Hemorrhagic*; 8th. *Corymbosa*.

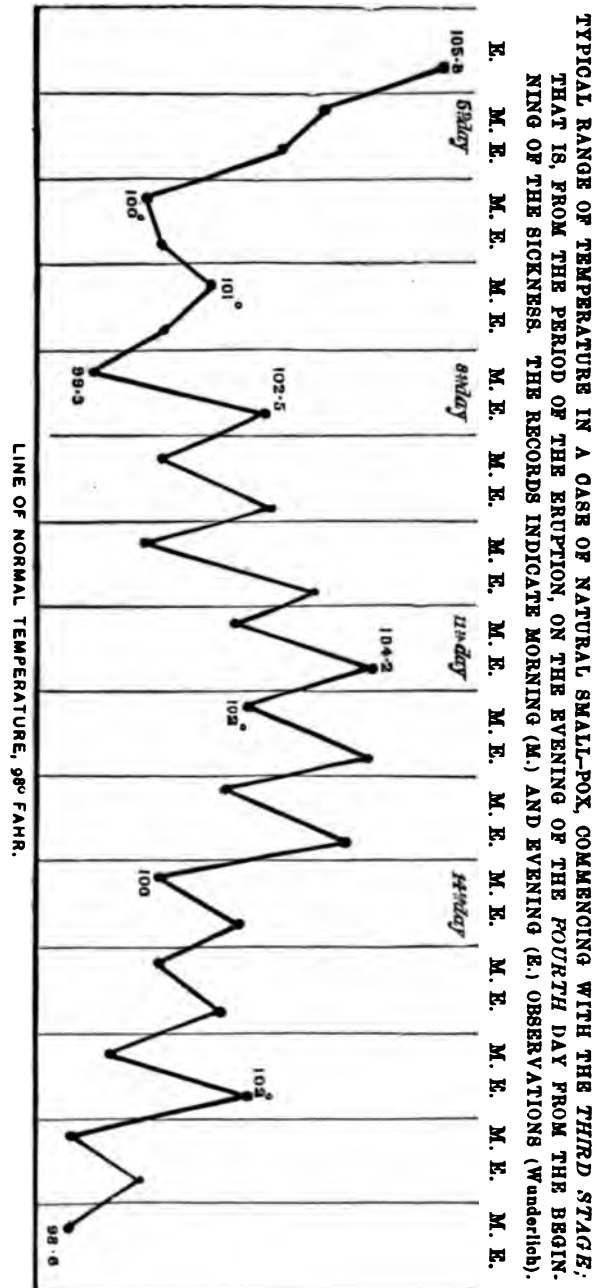
Symptoms of the Small-Pox without Eruption.—Sydenham and Frank observed in every variolous epidemic that some few persons who have not previously had the small-pox, or, according to Frank, have neither had the small-pox nor been vaccinated, are seized during the time the small-pox is raging, with all the symptoms of primary variolous fever, which having subsided, they have afterwards been found insusceptible of the disease. Sydenham states that he has seen fatal cases of this kind attended with purple spots and bloody

urine,—and hence the “*variolæ sine eruptione*” of authors,—which, when it occurs in the present day, is more usually regarded as a modification of small-pox, probably depending on the influence of vaccination.

Symptoms of the Distinct Small-Pox.—The symptoms of *variolæ discretæ*, or of distinct small-pox, may be divided into *four stages*. The *first stage* comprises the period of incubation or of latency—a period of time which varies according as the poison has been introduced by the mucous or cutaneous tissues. In the former case, or in natural small-pox, for example, the more usual time of latency is from ten to sixteen days; while in the inoculated small-pox the period of latency is from seven to nine days, the extremes, taking both forms of the disease, being from five to twenty-three days. Bärensprung, of Berlin, has lately recorded a most interesting fact, which demonstrates, in a more striking and definite manner, the period of latency; and which appears to be similar in persons who have been vaccinated and in those who have not. He observed seven cases of small-pox, *all* of which were infected from the same source on the same day. In *all* of them the outbreak occurred between the thirteenth and fourteenth day. Some of them were vaccinated and some were not (*Annalen des Charité Kranken*, vol. xix, p. 103). The *second stage* comprises the primary fever, which commences with the disease, and terminates with the appearance of the eruption. The *third stage* commences with the eruption, and terminates with the appearance of the secondary fever. The *fourth stage* commences with the secondary fever, and includes all the subsequent phenomena.

In the adult the symptoms of the second stage are mainly to be distinguished from those of the first stage of typhus, or other febrile affections, by the characteristic ranges of temperature. There is, however, a great tendency to vomiting, and to pain in the back, and the brain is oppressed, as indicated by drowsiness, stupor, or coma, followed occasionally by convulsions. The ordinary duration of this fever is four days, and it may be sudden in its attack, or be preceded by some days' illness, in which case the most prominent and characteristic symptoms in the adult are severe muscular pains simulating rheumatism, especially in the small of the back, and the frequent occurrence of obstinate vomiting, foreboding a severe form of the disease.

On the fourth day inclusive from the first attack of the primary fever, sometimes sooner, and but seldom later, the eruption appears, and the third stage commences. The phenomena of the third stage are as a calm succeeding to a storm; for, on the appearance of the eruption, the fever remits, the heat abates, the affection of the head subsides, the vomiting ceases, and the pulse returns to its natural standard, and consequently the febrile phenomena have altogether disappeared for the time. A temporary defervescence is thus well marked, the temperature falling, from perhaps 106° Fahr., progressively downwards to 100° Fahr.



The number of pustules varies, according to the severity of the case, from about twenty to some thousands. They appear first in minute bright-red specks on the face, neck, and upper extremities, then on the trunk, and lastly on the lower extremities, and run their course in a succession of crops. They undergo the various mutations of pimple, vesicle, and of pustule already described. About the eighth day of the disease, however, or when the eruption is fully out over the whole body, and the pustules on the face begin to mature, the whole face, head, and neck swell, particularly the eyelids, which often close and blind the patient; the swollen parts also throb, and are painful when touched. The intumescence of these parts lasts three days, during which the spaces between the pustules inflame, and are of a deep red or damask-rose color; and the closer this resemblance is seen to be, the milder will be the subsequent affections.

It is during this period of intumescence, simultaneously with the renewed hyperæmia of the skin, and introductory to the change taking place in the contents of the pustule, that the fever, which had remitted, returns, and the *fourth stage*, or that of secondary fever, commences—the *Fever of Suppuration*. This stage, in cases of ordinary intensity, is marked by a rise of temperature to a considerable height, by a frequent pulse, sometimes by a rigor, and by slight delirium, from which the patient is easily aroused. If, however, the disease be of greater intensity, hæmaturia, hæmoptysis, or a hard dry cough, are added. In favorable cases the swelling of the face, the redness of the intervening spaces, and also this secondary fever, having lasted from the eighth to the eleventh or twelfth day, subside, and the pustule, now fully ripe, bursts and discharges a thin yellow matter, which, concreting into a crust, falls off on the fourteenth or fifteenth day, and the disease terminates. During this somewhat protracted defervescence the temperature sinks gradually, to rise, perhaps, for the third time, when the desiccation takes place.

In the very mild variety of distinct small-pox which was wont to be named the "*horn-pox*," the primary fever is little more than a febricula; the pustules do not exceed half a dozen to two or three hundred; and, having passed through the stages of pimple and of vesicle, they, on the eighth day—*i. e.*, about the usual time of maturation—shrivel, desiccate, and crust. The secondary fever, often so fatal, does not recur, so that the convalescence usually commences on the eighth day, and the disease is terminated on the eleventh.

It was once supposed that in such cases the pus of the pustules was absorbed, but it appears that pus does not form, the fluid always remaining serous. In cases of any degree of severity, even in the *distinct small-pox*, the poison acts not only on the skin, but also on the buccal and conjunctival membranes, and produces an exudation on those parts. This additional affection, however, does not appear to aggravate the fever, or to occasion other inconvenience than what arises from the local disease. The buccal eruption is usually preceded and accompanied by soreness of the throat and difficulty of swallowing, and sometimes salivation; but these symptoms do not

exceed those of a common sore throat. The exudation upon the mucous membrane is generally resolved without the formation of ulcers, or anything that can be considered a scab or cicatrix. The exudations which form within the eyelids are not attended with much pain, and it is only when the swelling has subsided that the mischief which sometimes takes place is discovered.

A peculiar faint and sickly odor, of a "greasy, disagreeable" kind, and quite *sui generis*, emanates from the small-pox patient during the period of maturation of the pustules. So much is this the case, that Dr. Watson says "one might name the disease at once by the smell." When, however, the disease assumes an unfavorable character, and threatens a fatal termination, the face, which ought to have been intumescent on the eighth day, remains without increase of size, and the spaces which ought to have inflamed are pale and white. The pustules also, says Sydenham, look red, and continue elevated (even after death), and the saliva, which flowed freely up to this day, suddenly ceases. At this critical period the secondary fever, instead of its usual sthenic character, may assume one of two forms,—namely, either a form like the second stage of typhus, with brown tongue, frequent pulse, and delirium; or the patient may be overwhelmed with the depressing influence of the poison, and sink almost without experiencing a reaction, the pulse being hardly increased in frequency, the heat of the body natural, and the intellect unimpaired. But the patient suffers from an indescribable restlessness, an inexplicable anxiety, some cough, with sickness, a frequent desire to pass urine, and with these symptoms he dies.

Symptoms of the Confluent Small-Pox.—The confluent small-pox is described by Sydenham as beginning with symptoms similar to those of the distinct small-pox, but more violent; the second stage, or primary fever, being attended with more sickness and vomiting, with a higher temperature, with rigors, with more severe muscular pain, with more considerable delirium, and in children often, on the evening before the eruption, by convulsions. This fever is not only more intense than in the distinct kind, but is of shorter duration, and more tumultuous—the eruption appearing more generally on the third day, or even earlier; and the sooner the pustules appear, so much the more confluent is the disease that follows likely to be. The eruption is often preceded by an extensive erythematous or erysipelatous inflammation, and the pimples come out irregularly, or in small clusters, like measles, and are less eminent than in distinct small-pox. "The pustules run together over the greater part of the body."

When the third or eruptive stage is formed, the primary fever remits, but not so completely as in the distinct kind, for the pulse often continues frequent (110 to 120 in a minute), and the temperature does not fall so distinctly, the tongue is white, and even the delirium may recur in the evening. This eruption also has some remarkable characters; for the pustules, especially those of the face, do not rise; they are more irregular and flatter in their forms; and, from their greater number and contiguity, run into each other, or are confluent, sometimes forming bullæ as large as a hen's egg, and sometimes scarcely a portion of healthy skin is visible.

Other symptoms, sometimes seen in the distinct small-pox, never fail to accompany the second stage of confluent small-pox—namely, *sore throat* and *salivation*. The tonsils and the fauces become tumid and red, the face begins to swell, and then the salivary discharge begins either with the eruption or within a day or two afterwards. The discharge of saliva is at first thin and copious, resembling the ptyalism of mercury. About the eighth day, however, it becomes viscid, and is expectorated with difficulty; while in bad cases it either ceases for a day or two, and then returns, or it disappears altogether abruptly; and if the swelling of the face also subsides suddenly, the danger is great. Children are not so liable to this salivation as adults. In them, however, a vicarious diarrhœa often appears, but not constantly; neither does it occur so early in the disease. It is frequently profuse, and often proceeds till the disease terminates. Not unfrequently the larynx and trachea are implicated, even to the larger divisions of the bronchia. There is cough, with hoarseness, painful expectoration, and sometimes complete extinction of the voice. These are most dangerous symptoms.

It has been stated that, on the appearance of the eruption and the commencement of the third stage, although the fever is mitigated, it does not altogether subside, defervescence is incomplete, and the affection of the head, the frequency of the pulse, and greater heat of the surface, often continue. With these ominous symptoms still present, on the eighth day of the eruption, or the eleventh day of the fever, the fourth stage, or secondary fever, commences, bringing with it new sources of anxiety to the physician and of danger to the patient. Gregory and Watson both consider the eighth day of the eruption as the most perilous day of the disease. Blood often appears in the urine in slight and sometimes in large amount. Renal cylinders are not uncommon. The bladder is affected in a great number of cases, and there is increased mucus. If the urine be retained in torpid and semi-comatose cases, it becomes soon ammoniacal, as in all cases with catarrhal cystitis (Parkes *On the Urine*, p. 262).

"The confluent small-pox," says Sydenham, "does not in the least endanger life in the first days of the illness, unless there happens a flux of blood from the urinary passages, or from the lungs. Yet, on the decline of the disease, or on the eleventh, fourteenth, seventeenth, or twenty-first days, the patient is often brought to such a state that whether he will live or die is equally uncertain. He is first endangered on the eleventh day by a high fever (and the height of the temperature may indicate the danger), attended with great restlessness, and other symptoms which ordinarily prove destructive, unless prevented by medicine. But should the patient outlive this day, the fourteenth and seventeenth are to be apprehended, for a very vehement fit of restlessness comes on every day towards evening, and there is the greatest difficulty in saving him." The disease is apt to prove fatal by way of apnœa, after the eighth day; but after that period the characters of asthenia supervene.

The fatal symptoms of the fourth stage are, the absence of the usual redness in the intermediate spaces, the non-intumescence of

the face, the suppressed salivation, cough, with hæmoptysis or hæmaturia, and great restlessness. Sometimes other symptoms are added to these, as a brown tongue, delirium, petechiæ, or a black spot in the centre of each pock, scarcely so big as a pin's head; or a disposition to gangrene in the large vesicles. When these symptoms are present, few patients survive the crisis. In some cases, however, the event is favorable, and the patient is restored; but the struggle is sharp, and the convalescence long. In its progress an endless series of abscesses may form, or inflammation of a joint may take place, and produce lameness; ulceration of the cornea, blindness, otitis, or deafness may also ensue; while the deeply-scarred face is a lasting record of the severity of the disease, and of the great danger the patient has survived.

2. *Of the Inoculated Small-Pox.*

Symptoms.—The phenomena which result from the introduction of the variolous poison by means of the cutis differ in many respects from those that occur in the natural small-pox; and they are as follows: On the day after the operation is performed, little alteration is discovered in the punctured part. On the second day, however, if the part be viewed with a lens, and the operation has succeeded, there generally appears an orange-colored stain around the incision, while on the fourth or fifth day the part is hard, slightly inflamed, and itches, and a vesicle containing serum is formed on it. About the sixth day some pains and stiffness are felt in the axilla, symptoms which foretell the near approach of the fever and the favorable progress of the disease. On the seventh day the vesicle becomes more developed and the red areola forms round its base.

The operation having now been performed seven, eight, or nine days (the usual period of latency of the poison when so inoculated), and the vesicle having existed four days, the ordinary symptoms of primary fever appear. This fever lasts three or four days, when the general eruption follows, now called the secondary eruption, the pustules coming out, as usual, in three successive crops, on the face, trunk, and lower extremities. On the day of the general eruption the primary pustule, says Dr. Gregory, is distended with matter, and proceeds on its course, so that it has scabbed when the secondary eruption is only about to mature.

The most remarkable phenomena, however, of the inoculated small-pox are the singular mildness of the fever and the diminished number of the pustules of the secondary eruption. The mildness of the fever is thus instanced by the late Dr. Watson, of the London Foundling Hospital: "Of the seventy-four persons whose histories I have related, though inoculated with variolous matter in different states, although prepared in so different a manner, and a great number not otherwise prepared than by an abstinence from animal food, not one of them were disordered enough during the whole progress to occasion the least anxiety for the event; not one of them had their eyes closed a single day, from the pustules being

upon the eyes or near them ; none continued in bed an hour longer than they would have done in their best health."

The number of pustules is subject to great varieties, but, with very few exceptions, is much less than in the natural small-pox. In some cases not more than two or three appear ; occasionally only the primary pustule is seen ; but more generally the number varies from ten to two hundred, the mean being thirty or forty. Such is the general course of the inoculated small-pox. In a few instances, however, the disease that follows this operation is extremely severe, and in a still smaller number it is confluent ; and in either case the patient is perhaps destroyed. Many theories have been propounded to explain the singular mildness of the inoculated small-pox, but none of them are satisfactory.

Complications of Small-Pox and Special Morbid Tendencies.—Small-pox having been chiefly studied previous to any sound knowledge of morbid anatomy, or of morbid poisons, the occasional subsequent affections of the disease are still but imperfectly known. About the eighth day in the distinct small-pox, and the eleventh day in the confluent small-pox, a secondary fever is established, and at the same time a new series of phenomena may present themselves in a few severe cases,—as affections of the lungs, of the pleuræ, or of both ; of the urinary organs, or of the areolar tissue of the body generally. It is during the progress of this *secondary fever* that frequent opportunities occur for its degeneration into a fatal type. In such cases complete defervescence is never established ; but lesions become developed whose advent is capable of being appreciated by careful records of morning and evening temperature during the progress of this *fever of suppuration*. These are the *tertiary affections*, the eruptions and the fever being the *secondary effects* of the specific poison.

The most frequent affection of the lungs is hæmoptysis, but occasionally inflammation of these organs takes place, generally as pleuro-pneumonia. The mucous membrane, for instance, of the trachea is found often covered with a thick semi-purulent, muciform matter, peculiar to small-pox, irregular or honeycombed at its free surface, and which being removed, the subjacent tissue is found diffusely inflamed. The substance of the lungs also is occasionally found inflamed in every degree, even to purulent infiltration. The pleura also, according to Dr. Gregory, is peculiarly disposed to inflammation, which comes on about the eleventh or twelfth day, for the most part very suddenly, and proceeds rapidly to empyema, sometimes destroying the patient in thirty-six hours. The inflammation of the pleura does not merely run into suppuration, but takes every other form to which it is at any time liable.

The *tertiary action* of the variolous poison on the urinary and genital organs is seen in the frequent occurrence of hæmaturia, in the occasional formation of abscess of the kidney, in the occurrence of peripheric and parenchymatous orchitis, and in ovaritis ; while its action on the uterus is manifest from menorrhagia in the unimpregnated state, and by frequent miscarriage when the patient is parturient. The areolar tissue of the body generally is also acted

upon by this poison. In some cases examined a few hours after death the bodies can with difficulty be laid on the table, the skin being detached by the pressure necessary to raise them; and the serous coat of the intestines separates from the mucous and muscular coats with the greatest facility for many feet, and apparently might be entirely peeled off. In some cases the finger can be thrust through the walls of the heart with ease, as if the muscle of that organ had become unnaturally soft and broken down. This affection of the areolar tissue generally is seen in the great tendency to the formation of abscesses on the subsidence of the eruption; for twenty, thirty, and even more abscesses will sometimes form on a limb or other part of the body, in most formidable succession, and which, on being opened, are found to contain sanious, or, only in a few instances, laudable pus.

Pyogenic Fever.—In a case of septicæmia, occurring during the course of confluent small-pox, examined by Dr. Parkes, the disease ran its course well till the eleventh day, when there was shivering; on the following day there was bilious vomiting; on the fourteenth day there was sudden pain in the right wrist, and swelling of many joints; and on the following day there were all the well-marked symptoms of pyæmia. A daily examination of the urine showed the remarkable fact that the amount of sulphuric acid passed continued progressively to increase daily—rising from 23.8 grains to 44.4 grains (Parkes *On the Urine*, p. 267).

Sequelæ.—The different lesions that have been mentioned are not the only miseries from which the patient may suffer; for these are often followed by sequelæ even more formidable than the preceding phenomena, as blindness, deafness, or lameness. With respect to blindness, it is generally supposed that pustules form on the conjunctiva or cornea, the inflammation then extending to the deeper-seated parts, and thus destroying the eye. Mr. Marson, formerly surgeon to the Small-Pox Hospital, says that, according to his experience, "The eye seems to possess a complete immunity from the small-pox eruption, and that although it sometimes extends to the inner margins of the eyelids, the particular local affection that causes the destruction of the organ of vision in variola begins generally on the eleventh or twelfth day, or later, from the first appearance of the eruption, and when the pustules in every other part of the body are subsiding. It comes on after the secondary fever has commenced, with redness and slight pain in the part affected, and very soon an ulcer is formed, having its seat almost invariably at the margin of the cornea. This continues to spread with more or less rapidity, and the ulceration passes through the different layers of the cornea, until the aqueous humor escapes, or till the iris protrudes. In the worst cases there is usually hypopion, and when the matter is discharged, the crystalline lens and vitreous humor escape. In some instances the ulceration proceeds very rapidly: I have, more than once, seen the entire cornea swept away within forty-eight hours from the apparent commencement of the ulceration; and, what is singular, now and then the mischief goes on without the least pain to the patient, or his being aware that anything is amiss

with his eyes." Further, he calculates that in 1000 cases 26 had ophthalmia, or about 1 in 39; and of these 11 lost an eye each, or 1 in about 100.

The inflammation of the buccal membrane may extend to the Eustachian tube, causing suppuration of the ear, and sometimes permanent deafness. It may spread also to the glottis; and the patient has been known to die suffocated by effusion into the areolar tissue around it, causing occlusion of the aperture. Sometimes it has terminated in ulceration, with the loss of a portion of the nose, or in caries of the jaw-bone, or in enlargement of the glands of the neck.

The soreness of the fauces and tonsils is often associated with pustules on these parts; and the tongue, the roof of the mouth, the inside of the cheeks, the uvula, and the *velum palati* may be covered with an eruption like pustules; and it has been much disputed whether the eruption forms on any other part of the mucous membrane. As a general principle, it does not; but Martinet found, in a man that died on the eighth day, the rectum covered with what he supposed to be variolous pustules. Rostan has seen the alimentary canal garnished with pustules similar to those of the mouth, from the œsophagus to the rectum. Sir Gilbert Blane also met with pustules on the mucous membrane of the intestines in two persons who died in the West Indies; and Rayer has given a plate representing pustules on the mucous membrane of the trachea. Dr. Mead's experience has made him state that, "I myself have seen subjects in which the lungs, brain, liver, and intestines were thickly beset with pustules." Dr. Pitzholdt, in the *Morbid Anatomy of Small-Pox*, writes that he has seen the peritoneum covering the liver and the spleen presenting appearances which he felt justified in regarding as the product of small-pox.

The pustules which form on the mucous membrane of the intestine, however, have not been very distinctly studied either as to their course or phenomena. Rayer terms them *rudimentary* pustules; and Dr. Watson believes the statement, that such pustules exist, to be a mistake.

A case of small-pox recorded by Dr. George Patterson, of Edinburgh, was examined by one of the most learned and discriminating pathologists of the day, Professor W. T. Gairdner. He observed pustules on the mucous membrane of the colon, and pronounced them to be identical with the pustules on the skin (*Edinburgh Monthly Journal*, 1849, p. 549). Still it appears to be doubtful whether such eruption on the mucous membrane of the intestine is not the same as that seen in cholera cases, extending (as I have frequently seen it do in cases I examined in the hospitals at Scutari, in 1855) throughout the whole intestinal tract. The appearance of eruption in such cases is due to the solitary mucous glands, which are filled with exudation, not of a purulent kind, but having all the external appearance of pustules.

Such are the pathological phenomena which occasionally complicate small-pox. Death, however, not unfrequently anticipates their

action, and destroys the patient during the primary fever, and before any of them are set up.

3. *Of the Small-Pox after Vaccination—Varioloid, or Modified Small-Pox.*

Symptoms, Course, and Modifications.—It has been already noticed that during the epidemic prevalence of small-pox, even before vaccination was known, cases of small-pox occurred in a very modified form: such as the occurrence of variolous fever without the eruption (*variola sine variolis vel eruptione*), or the occurrence of small-pox in which the eruption continued vesicular (*the crystalline pock*), and, lastly, the occurrence of small-pox in which the vesicles dried up instead of becoming mature pustules, and known as stone-pock, horn-pock, wart-pock (*variola verrucosa vel cornea*). Modern pathology now regards these varieties as the result of the modifying influence of vaccination: and they may now be all described and classed under the common name *varioloid*. Comparative mildness of symptoms and course is their great characteristic, the pustules being "cut short in their development by vaccination or a previous attack of small-pox." There appears to be every variety in the nature of the modification, of which the principal are:

1. A fever of three days, without eruption, affecting people during variolous epidemics.

2. A high and severe fever, followed by a very mild eruption, sometimes only a single pock: the slight proportion which the amount of eruption bears to the severity of the preceding fever is perhaps the most marked characteristic of varioloid.

3. The occasional appearance of a scarlet efflorescence like that of scarlatina or roseola, preceding the appearance of the proper pimples, which occur as a very scanty crop.

4. In some rare instances the eruption is confluent, but does not advance beyond the development of a pimple or vesicle, and begins to dry on the fourth or fifth day of the eruption, forming a small hard tubercle, which soon disappears.

5. Sometimes the eruption is pimple, vesicle, and pustule, at one time in the same case.

6. Sometimes the eruption runs its regular course, but stops sooner, sometimes on the sixth or seventh day, instead of the eighth or ninth. In general, it may be stated that the severity and fully developed state of the disease is in proportion to the length of time which elapses from vaccination (COPLAND).

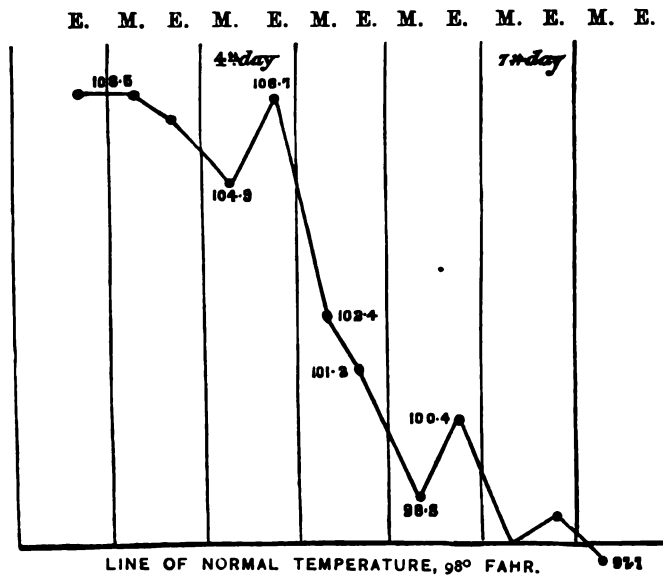
7. The varioloid eruption wants the peculiar odor of natural small-pox, and secondary fever is very rare.

8. Other eruptive affections—such as measles, scarlatina, purpura—materially modify the course and symptoms of small-pox.

Generally, it may be stated that, after an intense continuous fever, lasting a few days, a final exacerbation terminates the fever suddenly and simultaneously with the development of the small-pox pimples. A rapid and perfect defervescence then ensues, the temperature decreasing seven or even more degrees (Fahr.) within

thirty-six hours. From this event the patient remains entirely free from fever—*provided there exists no serious complication*—in spite of the continuous and progressive development of the small-pox pimples into pustules, and even in spite of the successive eruption of new pimples.

TYPICAL RANGE OF TEMPERATURE IN A CASE OF SMALL-POX MODIFIED BY VACCINATION. THE RECORDS INDICATE MORNING (M.) AND EVENING (E.) OBSERVATIONS, COMMENCING ON THE EVENING OF THE SECOND DAY (Wunderlich).



Exhaustion of Susceptibility.—The small-pox has the property, in common with measles and scarlet fever, of exhausting, on the first attack, the susceptibility of the constitution to the future actions of the poison. This law, however, is not without some exceptions, and in an epidemic at Marseilles, Bosquet considered that one person in one hundred was attacked a second time with small-pox. In some few instances even a second attack has no protective influence. Dr. Roupel says he met with an instance in which small-pox occurred three times in the same person. The lady of a Mr. Guinnett had it five times. Dr. Matson speaks of a lady who had it seven times; while Dr. Baron mentions a surgeon of the South Gloucestershire Militia who was so susceptible that he took small-pox every time he attended a patient laboring under that disease.

Coexistence of Small-Pox with other Morbid States.—The variolous poison is capable of *coexisting* with many other poisons, and also of influencing their actions, and of being reciprocally influenced by them. Dessessarz has seen variolæ coexist with scarlatina and with whooping-cough; Cruickshanks with measles; Frank with psora; and Dimsdale with syphilis. A patient was admitted into St.

Thomas's Hospital with tertian ague, writes Dr. Williams; the ague subsided and the small-pox appeared. The small-pox having run its course, the ague immediately returned. Ring mentions a case of triple disease coexisting—namely, small-pox, measles, and whooping-cough—and that they all ran their course together.

[An instance of the coexistence of small-pox and scarlet fever has been recently related by Dr. Sansom, of London (*British Medical Journal*, May, 1868). The case was a lady æt. 26, who, after suffering five days with the prefatory symptoms of scarlet fever, had the rash and throat symptoms, and at the same time there was violent pain in the back and loins. Seven days after the initial symptoms, papules of modified variola began to appear, and were developed in the ordinary way. On the ninth day from the commencement of the illness, a scarlatina rash was coexistent with small-pox eruption in the vesicular stage. Dr. Rugg, of London, has reported a similar instance.]

Cause.—The same obscurity hangs over the cause of small-pox as over that of many other diseases, such as of measles and of scarlatina. There is every probability, however, that these diseases have now no other mode of communication than from one person to another. There are some grounds for believing, however, that small-pox, in common with some other distempers, originated in the lower animals, and extended from them to the human species by infection or contagion. Sheep, we know, are liable to a distemper of the nature of small-pox; and there is every reason to infer that the disease is perpetuated by its own specific poison, miasm, effluvium, or virus, which spreads it about by the media of impalpable substances technically called "*fomites*," and which are capable of receiving, preserving, and carrying the germs of the disease. By such impalpable means the disease has been propagated since its first appearance in the world. The poisonous material of small-pox is given out from the mucous and cutaneous surfaces of a patient, especially from the lungs and skin, from the exhalations, the secretions, the excretions, the matters in the vesicles and pustules, and the scabs. These all contain the noxious germs of the disease, which may attach themselves to bed-clothes, body-clothes, and especially to woollen, cotton, and felted articles. Such stuffs retain the specific poison for a very long but undetermined period: any number of years, so far as is known—just as the hat, cap, and coat worn in a dissecting-room retain the peculiar effluvia of that place for a very long period.

It is not yet determined at what period this poison is first generated by the patient's person, whether during the primary fever, or not till after the eruption has appeared; but it probably begins to form and multiply during the primary fever. Generally, it may be stated that the poison is most powerful when it is most manifest to the sense of smell; that the dried crusts of the pustules or scabs possess the power of communicating the disease, and retain this power for a very long time. It is unsafe for a susceptible person—*i. e.*, a person who has not been vaccinated, or has not had small-

pox—to be in the same room, or in the same house, with a patient laboring under the disease. It has been caught by passing a child ill of small-pox in the street; so that “to expose a person in the public highway, infected with this contagion, is considered a common nuisance, and indictable as such.” The dead body of a variolated person is equally infectious, and students who have been near it when brought into the dissecting-room have in consequence had the disease communicated to them, although they may not have touched the body (CÆSAR HAWKINS). The *infecting distance*, therefore, must be many yards around the patient’s person: indeed, with every precaution, there is great difficulty in preventing it spreading from ward to ward in large hospitals during the prevalence of the disease. “There is no contagion so strong and sure as that of small-pox: none that operates at so great a distance” (WATSON).

The fact that small-pox is communicable has been fully demonstrated by the once general practice of inoculation. The poison by this operation has been proved to exist in the serum, in the pus, and in the crusts of the small-pox pustule. There is no law more singular and unexpected, in the whole range of morbid poisons, than that the introduction of the variolous poison, by means of the cutaneous tissue, should produce an infinitely milder disease than when the same poison is absorbed by a mucous tissue. Then the poison seems to be much more uncontrollable in its operations, as in the case when it affects a person who breathes an infected atmosphere with one who has been *inoculated* with the small-pox poison inserted beneath his cuticle through a puncture of the skin. Several explanations are put forward, namely,—(1.) That the small quantity of the poison conveyed by inoculation into the blood may make the difference; (2.) That the disease is milder when the poison is admitted through the cutaneous than through the mucous tissues; (3.) It may be held that in passing through the absorbent mucous membrane the poison is not only admitted in large quantity, but its potency may be increased and its amount multiplied by the living cells of the mucous membrane through which it passes.

The causes which predispose to small-pox or increase the susceptibility of infection are,—(1.) A very early age. (2.) Not having had the disease before. (3.) Not having been vaccinated: such are called “unprotected persons.” (4.) Peculiarity of constitution—*e. g.*, the Negro and dark races. (5.) Fear of infection. (6.) Epidemic influence.

It is gratifying to know that of recent years the prevalence and mortality of small-pox in this country is greatly less than was wont to be. Dr. Farr tells us that, for the three years previous to 1855, out of every 1000 deaths from all causes, only 7.607 were from small-pox.

Prognosis and Causes of Death.—The prognosis of the natural small-pox is always most grave. The danger may be measured, to a certain degree, by,—(1.) The quantity and confluence of the eruption; (2.) The state of the circulating fluids; (3.) The presence and nature of the complications, especially those of the respiratory organs and

nervous centres; (4.) Age, and habit of body of the patient; (5.) Nature of the epidemic constitution which may prevail.

Natural small-pox in unprotected persons is generally very fatal. The deaths average one in three. The fully formed confluent small-pox is always very dangerous. About one in ten die of distinct natural small-pox; and one to three per cent. only of small-pox after inoculation or after vaccination. The calculation of the proportionate number of deaths, however, appears to have greatly varied in different years.

There are certain signs regarded as unfavorable,—for example: excessive lumbar pains continuing; the persistence of vomiting after the appearance of the eruption; the occurrence of delirium, convulsions, or coma in adults during the primary fever; great confluence and simultaneous appearance of the eruption over the whole body. Such unfavorable signs are not necessarily fatal; but unfavorable signs which appear during secondary fever forebode, with greater probability, a fatal end. These are: the absence of the usual redness in the intermediate spaces; the distribution of petechiæ in the interstices; the development of a black spot hardly so large as a pin's head in the centre of each pustule; a livid or purple color of the pustule; a disposition to gangrene in the larger vesicles; imperfect development of the pustules, or their sudden subsidence, without remission of symptoms; sudden suppression of salivation; sudden suppression of urine; hæmaturia; cough with hæmoptoe; absence of swelling in the hands and feet when the eruption is copious; tendency to the formation of abscesses (*pyogenic fever*) after desquamation has commenced; congestive pneumonia, or bronchitis, with livid lips, face, or extremities, with hoarseness or complete aphonia. Recovery may take place even although the first-mentioned of these unfavorable signs exists; but convalescence is likely to be retarded by ulcerations of the cornea, asthenic ophthalmia, purulent deposits in the joints, ulceration of cartilages, otitis, abscesses and suppuration in the areolar tissue under the skin.

The development of scrofula and phthisis is apt to follow the disease, even though no unfavorable symptoms occur. In pregnant women the disease is always dangerous, often fatal, and almost always produces abortion; and the fetus so parted with not unfrequently bears evidence of small-pox upon the skin.

The most common causes of death are due to combinations of the unfavorable signs already noticed; and, according to Dr. Gregory's observations at the Small-Pox Hospital in 1828-29, the greatest number die on the eighth day of the eruption; or the eleventh day of the fever is the most fatal period. In private practice, Dr. Wood, of Philadelphia, considers the period between the twelfth and eighteenth day as the most dangerous to life. The greatest mortality from small-pox is in the early periods of life,—for example, before the fifth year. Dr. Farr estimates that out of every 100 deaths from small-pox, 75 are below that age.

Diagnosis.—It is not possible to distinguish, except by careful records of the temperature, the primary fever of small-pox from that incident to many other diseases with eruptions, or from the first

stage of continued fever. It is for the most part characterized by excitement rather than depression; and in the adult the muscular pains and pains in the back and loins are more severe and intense than in ordinary fever. The pain of the back is central in its position—a *spine-ache*—and is less affected by change of posture than the pain which is characteristic of lumbago, which affects the muscles at the side of the spine (often on one side only), and which is much aggravated by movement (BARCLAY). If vomiting occurs, which cannot be ascribed to any obvious cause, and persists till a papular eruption appears on the third or fourth day, with a remission of the febrile symptoms, little doubt can exist as to the variolous nature of the disease.

The diseases with which it may be, at first, confounded are, *petechial eruptions*, *measles*, and *chicken-pox*, and the *secondary pustular eruptions of syphilis*.

The nature of the fever, the character of the eruption, and the absence of any tendency to suppuration, are sufficient to distinguish *petechiæ* from variolous eruption.

Small-pox is to be distinguished from *measles* by the symptoms, as well as by the form and successive changes of the eruption. Crescentic patches, terminating in desquamation on the fourth day, characterize *measles*, as compared with small-pox, the eruption of which, even although it may be at first in efflorescent patches, never fails to become vesicular and pustular, proceeding to suppuration or blackening on the eighth day—a process which never fails to be attended by secondary fever.

It is more difficult to diagnose between varioloid and varicella, or chicken-pox. The chief difference consists in the eruption of chicken-pox presenting a vesicular character, which it retains; and it does not proceed to suppuration, but completes its course in five or six days, with a mild and short symptomatic fever.

The combination of mercury, scrofula, and syphilis often gives rise to cutaneous eruptions attended with fever, which may, in the first instance, be mistaken for small-pox. The eruption, however, is more tedious in its development, irregular in its course, and is persistent. It must, therefore, be distinguished by the history of the case, the long duration of the eruption, and the deep red or copper color it generally presents.

Treatment.—Since the first accounts by the Arabian physicians of the ravages of small-pox in Mecca, the history of this disease may be arranged in three great eras, each of which is characterized by remarkable epochs, and a fourth may be said to be now becoming apparent. The first of these eras is marked by an improvement in the treatment of small-pox. In few diseases has medical opinion undergone a more obviously beneficial change. To Sydenham is due the merit of this revolution in medical practice. The second era is marked by the discovery of the singular and beneficial phenomenon that the virulence of the poison of *small-pox* is greatly mitigated by introducing or ingrafting the disease into the system, through the cutaneous tissue, thereby causing the transference of the disease from one person to another, by inoculation. To Lady Mary Wortley

Montague is due the merit of having introduced the practice of inoculation into this country in 1722—a deed which must be considered as one of great heroism, when measured by the knowledge possessed by physicians in those days. The third great era is marked by the remarkable discovery which has rendered the name of Jenner immortal,—namely, the modifying and protecting influence of vaccination. He found that a certain disease in a cow, known as the *cow-pox*, could be transferred to the human subject by inoculation; and that, having been so transferred, it modified, to a considerable extent at least, the course of the disease, if it did not altogether prevent the occurrence of small-pox in its natural state in the human subject.

A fourth era may be said to have commenced in this country almost imperceptibly. It may be described as a period of transition, marked in this country by doubt and skepticism as to the efficacy of vaccination, tending to propagate an erroneous popular belief; and consequently, the ineffective adoption of means which practically have been proved to be sanative in the highest degree. In other countries, on the contrary, and especially in Central Europe, this period is marked by implicit faith in the virtues of vaccination, and the successful legal enforcement of this sanative measure.

An account of the treatment of small-pox resolves itself, therefore, into the consideration of two topics, namely,—(1.) The usual therapeutic curative, or sanative treatment of the disease; (2.) The sanitary treatment—*i. e.*, the means of protecting individuals from the *small-pox*; or of modifying the influence of the malady by inoculation or by vaccination. Of these in their order.

1. *Therapeutic, Curative, or Sanative Treatment of Small-Pox.*

The main object, in the first instance, is to prevent, if possible, a copious eruption; for the severity and danger of the disorder may be measured, in some degree, by this. The vulgar belief, that "better out than in," does not apply in the case of small-pox. The great object is to reserve the strength of the patient; and the attentions of an experienced nurse are demanded. A third indication is to watch for and deal vigorously with intercurrent inflammatory action, which is apt to be set up. The disease is not under the influence of any specific. It must run its course. But it is the business of the physician to assuage the untoward symptoms which may arise, by all the most approved methods of treatment, in accordance with the science of medicine of the present day.

Dietetic and General Treatment.—In the first instance, the course to be pursued is for the physician to act on the defensive, and simply protect his patient from certain injurious influences to which he may be exposed,—such as heating drinks to force out the eruption, which are apt to be given by ignorant and officious friends. Throughout the whole course of the disease, the diet should be strictly limited to slops, sago, arrow-root, and ripe fruits.

The chamber in which the patient lies should be cool, and freely ventilated. The bed-clothes should be light, the body linen daily

changed; and, when the disease is long, the patient's back should be often examined, to prevent sloughing. The scalp likewise should be examined, and, if full of pustules, the hair should be cut off, to prevent its matting. If the disease be diagnosed early, however, it is proper to shave the scalp, because the irritation which attends the suppuration of the pustules is thereby diminished, and cold may be more efficiently applied to the head, if necessary. In the early stage of the primary fever, in severe cases more especially, it is necessary to have the bowels well opened in the first instance, and to keep them regular by saline medicine. A cathartic pill, composed of the following ingredients, will be found to be efficient in most cases, especially if aided by a seidlitz powder, given six or eight hours after the pill:

Two grains of calomel, one grain of the compound extract of colocynth, one grain of gamboge, and one grain and a half of scammony, made consistent with a little aromatic oil.

The bowels must be daily attended to afterwards, and castor oil, or rhubarb, or magnesia, &c., may sometimes be required. Saline diaphoretics, in the form of James's powder; or the "aqua acetatis ammoniæ," to which a grain or two grains of tartar emetic has been added, so as to have $\frac{1}{4}$ th or $\frac{1}{8}$ th of a grain in every table-spoonful of the mixture, is an efficient and cooling diaphoretic. Spirit of nitric ether, or the nitrate of potass, may be added if required.

The surface of the body, over the hands, face, and feet, may be sponged several times a day with tepid water, with a view to relieve the intolerable itching; but caution is necessary to prevent exposure to cold. Cold-cream, or a liniment of olive oil, glycerine, and lime-water, smeared from time to time over the itching surface by means of a camel-hair pencil, may be found to afford relief; and chlorine lotions are highly spoken of by Eisenmann. With regard to the occurrence of convulsions in children, it is *not* found that opiates, as recommended by Sydenham and Cullen, are expedient. When the children are robust, or previously in good health, local bleedings, by means of one or two leeches to the temples, are more beneficial. Delirium, violent screaming, intolerance of light or sound, heat of head, all of which indicate a tendency to meningeal congestion, still more clearly warrant the application of leeches.

With regard to the propriety of bleeding (general) in adults, it is now well ascertained that it will neither eradicate the fever nor diminish the amount of the eruption. Bleeding is only warrantable if the pulse be full and strong, combined with evidence of inflammatory congestion in the lungs, liver, or brain.

When delirium, with restlessness, wakefulness, and a frequent pulse, is continuous, an opiate is indicated; and, combined with tartar emetic, is most advantageously given. A draught composed of thirty minims of the solution of muriate of morphia, with half a grain of tartar emetic, will be found beneficial in such conditions, and especially when given at bedtime.

Cooling drinks of lemon-juice, tamarinds, neutral effervescing

powders, are always agreeable to the patient, who ought also, for the sake of coolness, to be very lightly clothed. After the eruption has fully appeared, this is all which in ordinary cases requires to be done, and if, towards the tenth or eleventh day, there is much restlessness or sleeplessness, an opiate should be given.

When the febrile symptoms do not abate, as they ought, in the regular course of the disease, cathartics may be daily required to keep the bowels open. The most approved are the saline infusion of senna, or the black draught, the compound powder of jalap, combined with calomel and some aromatic powder, such as ginger. In this disease the bed-clothes ought frequently to be changed, and abundance of cool fresh air supplied to the apartment. When the state of the skin *alone* seems to keep up the febrile irritation, an antimonial opiate may allay irritation and procure sleep, after which a cathartic may be given with advantage in the morning.

In the complications which sometimes ensue, such as *inflammation of the throat and base of the tongue*, opiates are found to be injurious. The general treatment must be by cathartics or purgative clysters, if swallowing is difficult. In the other inflammations, however, opiates are of the greatest service, provided the symptoms be not of cerebral oppression; and local bloodletting is always to be preferred to general. In bronchitis, nauseating doses of antimony every hour sometimes procure relief; and if relief does not follow in the course of thirty to thirty-six hours, doses of calomel and opium ought to be given every second hour till three doses have been taken, each consisting of *two grains of calomel and half a grain of opium*. If the symptoms are not then relieved, this remedy need not be carried farther.

In the advanced stage of the secondary fever the strength of the system requires maintenance and support; because the abundant suppuration and extensive cutaneous irritation combine to exhaust the strength, as shown by the weakened pulse, the dark and dry tongue, blueness, paleness, or coldness of the extremities. Tonics, stimulants, and generally nutritious diet, are now called for. Quinine, mineral acids, malt liquors, especially the light bitter ales, wine, and even brandy, may be demanded. The diet should consist of milk, strong animal broths, eggs, raw or lightly boiled, according to the discretion of the physician, regulated by the digestive powers of the patient.

To prevent the face from being seamed, scarred, or "pitted" by the suppuration of the pustules, has taxed the ingenuity of physiologists and physicians. It has been stated that the influence of the atmospheric air is essential to the development of the pustules, and, accordingly, anything which would effectually exclude this influence would prevent the occurrence of a scar. But it is evident that the chance of scars can only be diminished by those means which are calculated to allay the general violence of the disease. When the eruption is severe, it is almost impossible to prevent the formation of "pits," because the depression results from the expulsion of a small slough; and the more mild the suppurative inflam-

mation can be rendered, so in proportion will the chance of "pitting" be diminished.

The local means adopted to prevent "pitting" may be shortly stated as follows ;

1. To open each individual pustule after suppuration has commenced.
2. To cauterize the pustule with nitrate of silver.
3. To employ both methods—that is, to open each of the pustules when it becomes vesicular, and introduce a strong solution of nitrate of silver into the cavity of the vesicle. At the end of a week scales fall off and no pit is left. Or lastly, to paint the face with a solution of nitrate of silver, in the proportion of one drachm of the nitrate to the ounce of water.
4. The application of a mercurial plaster, with the view of producing resolution of the *papulæ*. The preparation in use for this purpose at the Children's Hospital in Paris consists of 25 parts of mercurial ointment; 10 parts of yellow wax; 6 parts of black pitch.
5. Sulphur ointment applied several times a day.
6. Calamine mixed with olive oil, to form a coherent crust (BENNETT).
7. Tincture of iodine, painted over with a brush.
8. Saturated solution of gutta percha in chloroform (Drs. GRAVES and WALLACE).
9. To smear the face over with common olive oil.

All of these applications are for the most part applied to the face, the hands, and the arms only.

The severity and the mortality of small-pox has led many to think of means by which the disease might be completely extirpated. This leads us to consider—

2. *The Prophylactic, Sanitary, or Preventive Treatment of Small-Pox.*

Fifty years ago it was generally taught, among English physicians, that small-pox attacked the same individual only once in the course of life, and that its double occurrence in the same person was either very rare or next to impossible. The observations of Drs. Willan, John Thomson, Mr. Cross, Dr. Barnes, Dr. Craigie, and others since the time of these eminent physicians, lead to the following general conclusions:

1. Small-pox, though in general attacking the same individual only once during the course of life, may, however, affect him a second and even a third time.
2. This happens much more commonly when the first attack has been one of mild distinct small-pox than when it has been severe; and if the first attack has been one of confluent small-pox, it is rare for the same individual to have a second attack.
3. It is established by numerous observations, that an attack of any one of the varieties which have been named spurious small-pox or chicken-pox, by no means secures the same individual from an attack of confluent small-pox at a subsequent period.
4. Small-pox produced by inoculation does not necessarily secure the individual against an attack of small-pox induced in the natural way.

5. Every previous attack, however, of small-pox, whether natural or inoculated, exercises some modification on that which succeeds. This modification may be various in degree, from very slight and almost imperceptible to very conspicuous and remarkable. In this modification the symptoms of eruptive fever may be mild and of short duration; and the eruption may consist of vesicles or hard pustules, which disappear without suppuration.

6. The most powerful modifying agent on the course of small-pox is the action of the cow-pox on the constitution, or the disease produced by the application of vaccine lymph to the exposed skin. The specific disease so induced, in a large portion of cases, not only renders the individual less likely to be affected by the variolous effluvia, but if he is affected, changes very much the character of the disease which may supervene. Though the fever which precedes the eruption in cases of this class be similar in form and equal in degree to that by which the inoculated small-pox is attended, the eruption is either papuliform or tuberculated, without much surrounding inflammation. A similar eruption is produced when vaccine and variolous matter are inoculated at the same time in the same individual; or when a person who is exposed to the variolous contagion has been inoculated with vaccine lymph early enough to mitigate, but not wholly to supersede the eruption of small-pox. In such circumstances the vaccine lymph and variolous matter restrain and counteract the operation of each other on the system and on the skin. To these eruptions of modified small-pox the general name of *varioid* eruptions has been applied.

7. Cow-pox destroys the susceptibility to inoculate small-pox almost entirely; but the susceptibility to the natural disease, or that by inhalation, it does not entirely extinguish. This susceptibility, however, it diminishes in a much greater degree, and much more effectually, than inoculated small-pox does.

8. The susceptibility to second attacks of small-pox,* and attacks of small-pox after vaccination, is principally favored by the existence of an epidemic constitution of the atmosphere, and by the circumstance of early life, or the age below ten years. If no epidemic influence exists, the occurrence of second attacks of the disease may not be observed for a long series of years. But if, on the other hand, the atmosphere should possess or acquire an epidemic or variolous constitution, then neither the circumstance of a previous attack of small-pox, nor vaccination, can insure many of those under ten years of age, and not a few between that and thirty, from attacks of small-pox.

The preventive management of small-pox consists,—(1.) In the artificial production of the disease by *inoculation*, or *artificial variolation*; (2.) In the modifying and protective influence of *vaccination*.

Inoculation consists in the application of small-pox matter or virus to the surface of the corium, exposed by a puncture or scratch. The result is a local inflammation similar to small-pox, attended

* The average number of second attacks of small-pox seem to be one per cent. (R. Acad. of Med., Marseilles, 1828; and B. and F. Med.-Chir. Review, Jan., 1848, p. 74).

with an eruption and a fever, generally milder in form than small-pox acquired by breathing an atmosphere contaminated with the specific poison of the disease; and which thus passes through the mucous membrane to infect the blood. This is called the "natural way" of contracting small-pox; and the course of the disease so induced has been already noticed. For obvious reasons, the operation of *inoculating* the poison of small-pox has been rendered illegal in this country; and the practice of vaccination has been attempted to be enforced by law. What remains to be said about *inoculation* will be considered under the next topic.

COW-POX.

LATIN, *Vaccinia*; FRENCH, *Vaccine*; GERMAN, *Kuhpocken*; ITALIAN, *Vaccinia*.

Definition.—Cow-pox is the product of a specific and palpable morbid poison, which is reproduced and multiplied during the course of the malady in the cow or in the human being. After a definite period of incubation (from the time that the specific virus is artificially implanted, or communicated by impalpable emanations or effluvia in the "natural way"), specific pimples form upon some part of the skin, which pass through the stages of vesicle, pustule, scab, and desiccation. During the maturation of these specific pimples the adjoining lymphatic glands swell; a febrile state is induced, denoted by increase of temperature, constitutional disturbance of functions, acceleration of the pulse (which, to a certain extent, has been observed to continue persistent in some cases); and a general lichenous, roseolar, or vesicular eruption makes its appearance on the trunk of the limbs. The disease runs a definite course, affords immunity from another attack (for a considerable time at least), and exercises (during that period) a protective influence from human variola.

Pathology and Symptoms.—The importance of a comprehensive knowledge of the pathology of variolous diseases generally, and of cow-pox in particular, lies in the relations of this latter disease to small-pox and to vaccination. Dr. Jenner named the disease "*variola vaccina*," implying thereby that one genus at least of the animal creation is liable to a disease of a kindred nature with that which attacks man. The disease in the cow was observed to be generally mild; in man it was observed to be most pestilential and fatal. It was observed, also, that the disease was communicable from the cow to man, and that persons so affected were protected from subsequent attacks alike of small-pox and of cow-pox. Dr. Jenner believed that the two diseases were in reality identical. It has now been shown by unquestionable evidence that cattle and horses have for centuries been known to be affected with a species of small-pox or variola. Every different writer who has seen the disease has given it a similar name. Previous to 1745 it was known and described in Italy (FRACASTORIUS, LANCISI, RAMAZINI) as a malignant disease which destroyed cattle almost as extensively as small-pox did the human race. It was first observed in this country in 1745, and again in 1770 it appeared among the horned cattle with so much severity that His

Majesty George III, in his speech from the throne, at the opening of Parliament on the 9th of January of that year, called upon the Houses of Parliament to take the subject into their serious consideration. The disease continued with more or less violence till 1780, and it was no doubt the expiring embers of this epizootic which Dr. Jenner found in Gloucester, and made the basis of his investigations during that and subsequent years. Dr. Layard described the disease amongst the cattle in England, in that year, in a paper communicated to the Royal Society; and he mentions that inoculation from cow to cow was successfully practised, to mitigate the severity of the disease; just as Mr. Simonds, of the Veterinary College, London, in 1862, successfully practised inoculation of the variolous disease from sheep to sheep, or lamb to lamb, throughout the counties of Wilts, Hants, and Dorset, when *ovine small-pox* was epidemic. The great increase of mortality from small-pox among human beings which occurred during the latter part of the last century is a fact of some importance in connection with the epizootic disease; for at other times and places it has been observed that when the cattle were scourged by the variolous disease, mankind were in like manner great sufferers from a similar epidemic. In the interesting lectures "Introductory to the Study of Fever," by Dr. Andrew Anderson, of Glasgow, we are told that while small-pox was raging with great violence at St. Jago, on the west coast of New Granada—to which a town named David, in Chiriqui, was situated about sixty or seventy miles to leeward—a few days (four or five) before the disease appeared in this latter town the small-pox had attacked and destroyed many monkeys in the forest. Dying and dead monkeys were seen on the ground covered with the perfect pustules of small-pox; and several sick monkeys were seen on the trees, moping or moving about in a sickly manner. In the course of a fortnight one-half of the inhabitants of the town of David were stricken with small-pox (ANDERSON, p. 70).

It is also within the experience of many medical men, that during the prevalence of small-pox, cattle are apt to become affected with cow-pox. Horses, as well as cows and sheep, are liable to the affection; and the countries where the disease of late years has been found are those where it has formerly been known to have existed among cows or horses in its most virulent form. During the epidemics of small-pox previous to 1840 the variolous affections among the cows of the country were more observed than at any period for many years. In the dairies of Suffolk, of Gloucestershire, Dorsetshire, and Buckinghamshire, the disease has prevailed not only during epidemic small-pox, but when no cases of variola were known to exist in the immediate neighborhood. There are good grounds for the belief that the impalpable emanations—the specific effluvia—from cases of small-pox in human beings, have been sufficient to communicate the variolous disease of cows. Mr. Ceely gives a most interesting history of such an occurrence in the tenth volume of the *Transactions of the Provincial Medical and Surgical Association*. At the village of Oakley, about sixteen miles from the town of Aylesbury, small-pox had been epidemic from June to

October, 1840. Two cottages, in which three persons resided during their illness, were situated on each side of a long narrow meadow, comprising scarcely two acres of pasture-land. One of these three patients, though thickly covered with pustules of small-pox, was not confined to her bed after the full development of the eruption; but frequently crossed the meadow to visit the other patients—a woman and child—the former of whom was in great danger, from the confluent malignant form of the disease, and died. According to custom, she was buried the same evening; but the intercourse between the cottages across the meadow was still continued. On the day following death the wearing apparel of the deceased, the bed-clothes and bedding of both patients, were exposed for purification on the hedges bounding the meadow; the chaff of the child's bed was thrown into the ditch; and the flock of the deceased woman's bed was strewed about on the grass over the meadow, where it was exposed and turned every night, and for several hours during the day. This purification of the clothes continued for eleven days. At that time eight milch cows and two young heifers (sturks) were turned into this meadow to graze. They entered it every morning for this purpose, and were driven from it every afternoon. Whenever the cows quitted the meadow the infected articles were again exposed on the hedges, and the flock of the bed was spread out on the grass, and repeatedly turned. These things remained till the morning, when the cows were re-admitted, and the contaminated articles were supposed to be withdrawn. It appears, however, that the removal of the infected articles was not always accomplished so punctually as had been enjoined, so that, on one occasion at least, the cows were seen in the midst of them, and licking up the flock of the bed which lay on the grass. These cows were in perfect health when first put out to graze in this meadow; but in twelve or fourteen days *five* (out of the eight) milch cows appeared to have heat and tenderness of the teats. The teats became swollen, and small hard pimples could be distinctly felt upon them, as if imbedded in the skin. These pimples daily increased in magnitude and tenderness; and in a week or ten days they rose into *blisters* (vesicles), passing into brown or blackish scabs. When the teats were in this condition, and very tender, constitutional symptoms of ill-health became developed. Sudden *sinking* or loss of milk, drivelling of saliva from the mouth, frequent inflation and retraction of the cheeks, staring of the coat, "tucking up of the limbs," "sticking up of the back," and rapid loss of flesh were the appearances which even the peasants themselves were able to appreciate. By the middle of the third week the pustules were mature, and the crust and loose cuticle began to be detached. The simultaneous occurrence of the disease on all the animals increases the probability of the operation of one common cause. The whole of the cows were certainly affected within less than three days of each other; and another circumstance requires particular notice, namely, the occurrence of the disease in a young heifer (sturk), to which of course the disease could not have been communicated by those casualties which commonly propagate the vaccine variola amongst

milch cows. The cause which originated the disease amongst them at the same time affected the young heifer, which hitherto had not been considered liable to the vaccine disease, simply because no one had seen the animal affected by it. Now it is known, both in this country and in Germany, to be liable to the disease.

The proprietor of the animals referred to in this narrative had the disease communicated to himself. He had never suffered from small-pox nor the vaccine disease; and it was his own spontaneous conviction "*that his cows had been infected from human small-pox effluvia,*" to which undoubtedly they had been exposed. He had not the remotest idea of the medical theories concerning the nature of the disease, and consequently had no prepossession in favor of the opinion he thus spontaneously expressed. His cattle had hitherto been in good health, and no vaccine variola had been known in the vicinity.

Human small-pox has also been communicated to the cow by direct implantation of the specific virus from man. The efforts at first were numerous and unsatisfactory to inoculate directly the cow with human small-pox; and the experiment is said to have first succeeded at the Veterinary College in Berlin, so early as 1801. M. Viborg, of Copenhagen, about the same period communicated the disease to dogs, apes, and swine. In 1807 Gassner imparted the small-pox to the cow by inoculation. In 1830 or 1831 Dr. Sonderland, of Bemen, communicated the disease to cows, by simply covering the animals with sheets and blankets on which persons suffering from small-pox had lain. In 1836 Dr. Basil Thiele, of Kasan, in South Russia, successfully inoculated some cows on the udder with the *virus* of human small-pox. Vesicles were produced bearing all the characters of the true vaccine vesicle in those animals. The lymph so produced from the *variola* of the cow continued to retain the specific properties of the vaccine variola throughout seventy-five successive transmissions in the human subject. In 1838 M. Thiele repeated this interesting experiment with a similar success. It would therefore seem that the constitution of the cow has the power of assimilating, of modifying, and of mitigating the human *variola* virus, and of stamping it with the properties of the vaccine variola. Dr. Ceely, of Aylesbury, twice succeeded in accomplishing this object (so important pathologically), after many fruitless trials. The interesting papers by him in the eighth and tenth volumes, and the Reports of the Vaccination Section of the Provincial (now British) Medical Association in their *Proceedings* for 1839 and 1842, should be studied by every student of Medicine. The main points of the statement here given are taken from these sources. Very recently (1860) Martin inoculated some variolous matter taken from a pock upon the body of a man who died of variola, into a cow's udder, and subsequently vaccinated about fifty persons with the matter derived from the cow. Most of those so inoculated were attacked with variola, and three died (*Boston Med. Journal*, 1860; *New Syden. Society Year-Book* for 1860). It would have been better, or at least more judicious, to have chosen a milder case than a fatal one to have inoculated from. Mr. Ceely has also often re-

communicated the vaccine disease from man back to the cow (*retrovaccination*, as it has been called); and he has observed that good human lymph, when *re-transmitted* in this manner, loses some portion of its activity. The phenomena appear later, small vesicles are produced, but ultimately, *after successive re-inoculations on man*, it regains its activity. Human small-pox has also been transmitted through the horse to the cow, and so to the child in the form of cow-pox (FLETCHER).

As the first origin of these specific poisons is as yet unknown, it cannot be now definitively determined whether man first had the disease communicated to him from the animal creation, or whether the lower animals, such as horses or oxen, had the disease communicated to them from man. The existence of small-pox in man is recorded in China as early as 1122 years before Christ (MOORE). And it is certain that when variolous disease appears among the lower animals in a malignant form, it is capable of producing, by inoculation, a disease of similar severity in man, if he has not already suffered from a similar affection; and that the direct inoculation of the cow with human small-pox produces a mild and mitigated form of disease—that such disease being again reproduced in man by inoculation from the mitigated disease of the cow, accords entirely in its character, in its progress, and in its protecting influence with the *variola vaccinae*, as described by Dr. Jenner. These and similar facts seem to lead to the conclusion that small-pox and cow-pox are not dissimilar diseases, but are identical in their nature.

There are some remarkable circumstances which must at once arrest the attention of the student, who carefully studies the accounts given of the experiments on men and animals, from which many of these statements are deduced. *First*, There seems to have been great uncertainty and difficulty often attending the actual attempts to transfer the specific virus of these eruptive or variolous diseases from one animal to another. The very interesting experiments of Ceely, and of Thiele and others, demonstrate this in a remarkable manner. *Second*, These experiments show the *marked improvement which sometimes takes place in the energy, and therefore in the quality of the specific virus, by subsequent removes or inoculations, in animals of the same kind, after the virus had been successfully implanted in one of them*. This energy and improved quality was shown in the more perfect development of vesicles, and in the more active manifestations of the primary and secondary symptoms. The subsequent inoculations of such improved lymph seem to produce less severe and less dangerous *local* results—the virus seems less acrid, less virulent, and less mischievous—having apparently acquired increased specific activity combined with mildness of action, and a greater susceptibility of transmission from one animal to another of the same kind.

Keeping, therefore, these facts in view, the history of the remarkable epizootic of *variola ovinae* which made its appearance in August, 1862, in some of the largest flocks of sheep in the West of England, is of great interest to the Pathologist. This variolous disease of

the sheep allies itself very closely with small-pox in man, with cow-pox amongst cattle, and with the vesicular eruptive diseases of the horse, but undoubtedly modified by the constitution of the sheep, just as the variola of man is known to be modified or transformed by the constitution of the cow into the *variola vaccina* of that animal.

The ovine variola is known as the *clavelée* of the French; and although this kind of *rot* was not observed in this country till 1847, when it was imported from Spain, yet it is a disease by no means uncommon as an epizootic in the flocks of Italy, France, and Moravia. In 1830 the mortality was considerable in Moravia; but by a timely inoculation with the *virus* of the disease, the remaining part of the affected herds were preserved. The artificially affected animals seemed to pass through a milder disease. To this kind of inoculation the name of "*clavelization*" has been given, from *clavelée*, the French word for the *tag-sore* or *rot*. This variolous disease in sheep assumes one of two forms, namely,—(1.) A virulent or malignant form; and (2.) A benign form. The virulent form (which would seem to have been the form epizootic in England) never produces pustules; and specific virus for safe inoculation (*clavelization*) can only be got from the benign form of the disease. When the disease is virulent, the sheep lose their eyes, their wool falls off, and their skin cracks in a zigzag manner. Their nostrils are so full of a fetid discharge that the shepherds are under the necessity of constantly syringing them with medicated lotions, to prevent suffocation. When the disease is benign, genuine pustules form, and every pustule, after the scab falls off, leaves a cicatrix in the form of a pit. On this cicatrix the wool never grows again. Hence it can always be told what sheep have undergone the variolous disease, as easily as it can be seen that a human being has had small-pox.

The prevention or mitigation of this disease among sheep is a most important object in a sanitary point of view.

In 1803 Dr. De Carro, of Vienna, tried the effects of the inoculation of *variola vaccina*, but without success. The inoculation only produced small local sores. It is said, also, that this sheep-pox cannot be communicated directly to the cow, nor to children (CEELY, SIMONDS). Other observers state, however, that it is so communicable, and that *ovination* is protective against small-pox (SACCHO). The *ovination*, or inoculation of the disease from sheep to sheep, was first proposed by Chalette in 1762, and has been yearly practised since that time in many parts of Italy, Prussia, Austria, and France. The practice of inoculation from sheep to sheep was practised in England by Professor Simonds during the recent epidemic. Results accrue to sheep, from the communication of the disease to them by inoculation, not less beneficial (compared with the fatal effects which followed when they became affected with the disease in the "*natural way*") than the beneficial effects that accrued to man when small-pox was communicated to him by inoculation—as it *rightly* was—before the protective powers of vaccination were known.

The ovine variola has been ascribed by some farmers to the communication of the virus from an eruptive disease of the horse (De

CARRO, RING). Fontan relates that some mares being affected with a pustular eruption, the matter from the pustules was inoculated on the teat of a cow, where it produced several fine pustules. From these several infants were vaccinated, with the result of producing perfectly characteristic vaccine vesicles. Thirty infants have been vaccinated from this source at Toulouse, and in all the result has been most satisfactory (*L'Union Méd.*, 1860; *New Syden. Society Year-Book*, 1860). If this can be definitely established, then the successful inoculation of some animal, *other than the sheep*, with the virus from the specific eruptive disease of the horse may give such energy and, at the same time, mildness to the morbid poison, by subsequent removes, that the implantation of the new virus (*equination*) may perhaps be followed by the same beneficial results to sheep, in respect of the malignant variolous disease to which they are liable, that *vaccination* has conferred on man in respect of small-pox. The question, then, at once suggests itself: "Has human small-pox ever been communicated to sheep, with the view of obtaining a modified lymph which may confer protection on them from the variolous disease to which they are liable?" From analogy, may we not indulge the hope that the practice of inoculating sheep from the small-pox of man might induce as mild and modified a disease in them, and prove as protective to them, as vaccine variola, through vaccination, has been to man? Or, having communicated the human variola to cows (as the experiments of Ceely and Thiele demonstrate that such may be effected), might not sheep be tried with the resulting virus as a protective agent? The vesicular eruptive diseases of dogs, as well as of horses, should be similarly inquired into and experimented with, seeing that dogs are so much associated with sheep.

The outbreaks of the variolous diseases amongst cattle and sheep seem to follow similar inexplicable paths to those which small-pox amongst human beings is observed to follow. Occasionally the disease is *epizootic* (equivalent to *epidemic* amongst men), or prevalent at the same time in several farms at no great distance. Cases spring up like small-pox, now and then, which appear to be solitary, and the source of which cannot be traced. It is rare indeed that the solitary cases of small-pox in human beings can be traced to a communicating source. In oxen it may be seen sometimes at contiguous farms; at other times, one or two farms, apparently similarly circumstanced amidst the prevailing disease, entirely escape its visitation. Sometimes it is introduced into a dairy by recently purchased cows. On the other hand it has been undoubtedly communicated to cows from the vesicular disease of the horse, through the hands of the common attendant on both animals. There can be no doubt, also, that the disease often exists, although it is not observed; for the disease being mild, and the tempers of the animals good, little notice is taken of tenderness in milking, and so the existence of disease escapes detection.

There are *spurious* forms of the disease, which it is very necessary to be able to distinguish.

In the true cow-pox there is very slight manifestation of fever or

constitutional disturbance. The secretion of milk may diminish ; but the animal continues to feed and to graze very much as usual. The local affection may be so mild that a single vesicle only may appear upon the udder of the cow ; but where the udder is voluminous, flabby, and pendulous, and uncovered with hair, with a corrugated, thin, or fissured skin, then there is apt to be a copious eruption. The disease is very readily propagated from cow to cow by the milkers ; and it is also said to be communicated in the natural way.

The local symptoms of the natural disease are evinced by heat and tenderness of the teats and udder for three or four days, followed by irregularity and pimply hardness of the surface, especially about the bases of the teats and the adjoining part of the udder. The pimples assume a red hue when about the size of a vetch or pea, and are quite hard. In three or four days more they increase to the size of a horse-bean, milking becomes painful to the animal, and the pimples become vesicles, which are then apt to be broken by the hands of the milkers, giving rise to troublesome and dangerous sores on the udder and teats of the cow, and communicating the disease to the milker, if he is not already protected by having had the disease before. If the vesicle remains unbroken it becomes a globular, oval, and ultimately a pointed (acuminated) pustule. A central depression, with a marginal induration, is the form ultimately assumed ; and when punctured towards the centre, the vesicles yield a more or less viscid amber-colored fluid. Dark-brown or black, solid, uniform crusts eventually form on the site of the vesicles. Some of these crusts may be seen semi-detached, others entirely so, and exposing a raw surface with a slight central slough. The forms of the crusts are either circular or oval, some flatter, and others unguiform, some thin, and more or less translucent. These varied appearances are seen in all stages at the same time, indicating the formation of new crops of vesicles at different periods. The period of incubation, after casual communication of the disease, seems to be from six to nine days, although it is said pimples may be felt under the cuticle about the fifth day. When the vesicles are fully mature, they may measure from eight to ten lines in the largest diameter ; the centre and edges of the intumescent margin being of a deep blue or slate color, and the surrounding areola of a pale rose color, and seldom more than four or five lines in depth, the integuments under it being deeply indurated. The lymph contained in the vesicle is now so copious that the cuticle over the central depression appears raised up by it, and so gives rise to a globular or cone-like vesicle ; or it spontaneously ruptures, when the lymph freely flows out, and concretes into a clear amber-colored crust or scab.

If undisturbed, this crust or scab gradually becomes thicker, darker, and more compact, till the thirteenth or fourteenth day, and spontaneously separates about the twentieth or twenty-third day. A cicatrix or pit is thus left, which is shallow, smooth, oval, or circular, of a pale rose or whitish color, with some traces of induration surrounding it. The anatomical structure of the vesicle seems to be precisely similar to that of small-pox in man, as shown now by many

observers (GENDRIN, CEELY, and others). The cow—like children and the young of other animals, particularly high-bred dogs—is subject to a purely vesicular eruption, which makes its appearance about the ninth or tenth day of the vaccine disease. The vesicles of this eruption, within twenty-four hours, contain a pellucid serous fluid, raising the epidermis. On the following day they become turbid, the cuticle collapses or bursts, and a thin, brittle, flimsy crust forms, and speedily falls off. Successive crops continue to form and desiccate for three or four weeks.

Primary Vaccine Lymph.—To procure *primary* liquid vaccine lymph direct from the cow, in a condition fit for use, is a task of no ordinary difficulty. *Primary* crusts should be sought for on the lower part of the udder and around the base of the teats; and during a search for these it is not improbable that smaller vesicles of later growth may be found to yield efficient lymph. The best lymph is to be obtained from perfect vesicles, before they begin to point. After this period it is less to be depended on, particularly if very abundant, thin, or discolored. Pointed vesicles, when broken by violence, are rarely to be relied on. Entire unpointed vesicles, or vesicles with central crusts, should be sought for on parts where they are least exposed to injury—namely, on the lower and naked parts of the udder and adjoining bases of the teats. It is impossible to exercise too much delicacy in the proceeding. The puncture to liberate the lymph should be made with a sharp lancet as *near the centre* of the vesicle as possible; and the epidermis may be gently raised to a moderate extent around the discolored or most depressed part. Slight pressure with the blade of the lancet, or between the thumb and finger, will enable the operator to charge a few points or capillary tubes with the slowly exuding lymph. Punctures at the elevated and indurated margin of the vesicle are utterly useless. They only give vent to blood. Vesicles on which the central crust has begun to form are the most productive, particularly if the crust be small and the margin of the vesicle be tender, hot, and tumid; and small superficial vesicles are often more yielding than contiguous larger vesicles, which are more deeply seated or confluent.

Useful substitutes for liquid lymph, capable of communicating the vaccine disease, are—(1.) Amorphous masses of concrete lymph, found upon or in close proximity to broken vesicles. They ought to be colorless, like crystals of white sugar-candy; or of a light amber hue, resembling fragments of barley-sugar. (2.) Central crusts, irregular, rough, and more or less conical; the more transparent and nearer a dark-brown hue the better. (3.) Vesicular crusts or desiccated vesicles. These crusts should be carefully removed by the milkers before they are casually removed or spontaneously fall; and those only of primary formation, which are as it were the mould of a vesicle, of a dark-brown translucent appearance, should be retained. These three dry conditions of the specific vaccine virus may be reduced to a liquid state at any time for use. Glycerine is said to be the best solvent for such solid conditions of the lymph, which ought to be reduced to powder before the glycerine is added (Collins, *Boston Med. and Surg. Journal*, 1858).

Vaccination.—It is now (1864) at least sixty-four years since Jenner first promulgated his discovery to the world, that the eruptive vesicular disease which has now been described as occurring on the udders and teats of the cow, and which he named the *cow-pox*, was communicable directly to the human being, and thus conferred protection from the small-pox, so fatal to man. The operation for thus ingrafting the cow-pox on the human being has been named "VACCINATION," and its discovery still remains one of the most interesting facts in the history of Medical Science. When an obscure apprentice with a surgeon at Sodbury, near Bristol, Dr. Jenner first caught a glimpse of this great truth, which he thoroughly investigated amongst the expiring embers of that epizootic disease which laid waste the herds of this country towards the end of the last century. He did not suffer the spark to be lost in the flame it had served to kindle. Amongst the gossip of the cow-herds he had heard of the vague, obscure, but popular belief regarding the possible communication of cow-pox to the milkers of the cows, and the protection from small-pox which the cow-pox conferred—a belief which undoubtedly prevailed in the rural districts of Gloucestershire, and, at the same time, curiously enough, on the continent of Europe, in some districts round Göttingen. These things Jenner mentioned to the famous John Hunter, at the time he was an apprentice to that eminent surgeon; but John Hunter, otherwise sagacious and far-sighted, pooh-poohed the notion as vague and improbable. Nevertheless, Jenner had determined to examine into the truth of the tradition; and he commenced his earnest and painstaking investigations as soon as he had established himself as a surgeon at Berkeley, in Gloucestershire. In June, 1798, he published his observations in the form of a thin quarto, of scarcely more than seventy pages, dedicating it to his friend, the late celebrated Dr. Parry, of Bath. Jenner seems to have felt almost a holy reliance in the truth of his great discovery; and in the face of much foolish opposition he modestly continued to prosecute his inquiry, "encouraged," as he said, "by the hope of its becoming beneficial to mankind." Its importance to the welfare of the human race has since been clearly demonstrated; and the acute observation of Jenner himself has been abundantly fulfilled—namely, that the keenest of all arguments for or against the practice of vaccination will be *those which are engraved with the point of the lancet*. We have, indeed, in this country, paid but tardy homage to his memory; nevertheless, he has imprinted for himself imperishable "footprints on the sands of time," which wave after wave of scientific research appears only to make more distinct. He has not only pointed out the means of subduing a loathsome disease, but the health of all civilized communities has improved, and, in proportion as vaccination has been efficiently carried on, the frequency of epidemics has been diminished, and the duration of human life has been extended.

The subject of vaccination is one which demands a careful study, alike in its pathological and in its sanitary relations. Questions of great national importance are concerned; and the following account

is mainly given from a notice of the subject, written by the author, in the pages of the *Medico-Chirurgical Review* for 1857:

In 1841 the Vaccination Act was passed, which rightly made the practice of *inoculation unlawful*. In 1853 another Act was passed, with the view of rendering the practice of *vaccination compulsory*,—an Act which is known as Lord Lyttleton's Vaccination Act. During the interval between the first and second reading of the Bill in the House of Commons, "The Small-Pox and Vaccination Committee of the Epidemiological Society completed a report on the prevalence and mortality of small-pox, and of the means taken to guard against it through vaccination." The conclusions they arrived at were deduced from the largest and most accurate mass of statistical evidence which had ever been brought to bear upon the question, and were eminently calculated to encourage Her Majesty's Ministers to pass an efficient measure to *compel* vaccination. A most valuable pamphlet was afterwards published by Dr. Seaton, which demonstrates the truth in a still more forcible manner, as to the protecting and modifying influence of vaccination in small-pox. To this belief, indeed, the general assent of the medical profession appears to have been given at least fifty years ago. Then, it would seem to have been all but unanimous; and now, one would think, at first sight, that it were almost an insult to human understanding to be obliged to collect statistics to *prove* that vaccination confers a large exemption from attacks of small-pox, and almost absolute security against death from that disease. But so it is, and independently of the information which such statistical inquiry is calculated to convey to those who advise our Lawgivers and Public Administrators, the inquiry is eminently useful in relation to everything which bears on the nature of vaccine and variolous disease. The general ignorance of the community, especially of the lower orders, as to the aim and object of vaccination, is lamentably great, and has still to be overcome. Moreover, the highest medical authorities of late years recommend that all views and facts put forward as objections to vaccination should be vigorously inquired into, and that there should be published from time to time a true account of such inquiries, with an elucidation of what has seemed doubtful and contradictory (SIGMUND, ALISON).

It is now well known that Lord Lyttleton's Vaccination Act (1853) has proved but a very imperfect measure—a piece of legislation which has fallen very far short of accomplishing all that is yet required. The inefficiency and imperfect working of the Act has been fully shown,—(1.) In the Reports of the Registrar-General for 1854; (2.) By the medical profession generally; (3.) By the medical registrars in particular; (4.) By the public, as expressed now and again in the newspapers of the day. To this state of things we owe a most valuable work on vaccination, written by the indefatigable medical officer (JOHN SIMON, F.R.S.) of the then (1857) General Board of Health. The aim of this publication was to lay before the Board such medical facts and considerations as might assist in estimating the hygienic value of vaccination, and the strength of any objections which may have been alleged against its general adoption.

This work is especially valuable, because it brings together a body of evidence down to the day of its publication—evidence of a pathological and statistical kind—such evidence as Jenner would have rejoiced to see—records which have been engraved by the lancet's point. But evidence of the inefficiency of the Vaccination Act of 1853 still continues to be apparent, as may be seen from the yearly reports of the medical officer of the Privy Council. During three or four years previous to 1860, Mr. Simon writes, that "Sometimes in one set of places, and sometimes in another, there have been occurring, almost generally throughout England, epidemics of small-pox more or less considerable." To such an extent has this been the case, that, in 1860, the Lords of Her Majesty's Most Honorable Privy Council deemed it necessary, with reference to local outbreaks of this disease, to enter into correspondence with the authorities responsible for public vaccination in several unions of England where small-pox had been prevalent. Some of these districts were specially visited by competent medical officers, and in two adjoining Devonshire Unions it was ascertained that the *diffusion of small-pox* had actually been to some extent *wilfully promoted by the illegal practice of INOCULATION!* Alarm was of course justly excited among educated persons in the endangered places, by the knowledge that this offence was being committed; and in one case, where there was reason to believe that *inoculation had been the cause of death*, Secretary Sir George C. Lewis offered a reward of £50, to be paid to any person not actually concerned in the offence, who would give information and evidence leading to a conviction of the offender.

The unsatisfactory working of the Vaccination Act of 1853, now amply proven, has led to the promulgation of an Order of the Privy Council (of date December 1, 1859), for the improvement of public vaccination. Their Lordships have seen fit to direct the commencement of a *systematic inspection*, with reference especially to the operation of vaccination, and its efficiency in Unions where the amount of infantine vaccinations, compared with the number of births, appeared to be especially low. These inquiries continue to show that the present law, "to extend and make compulsory the practice of vaccination," is so imperfect as to be almost inoperative; and the *systematic inspections* instituted seem to have been so useful in promoting vaccinations that their Lordships propose to continue them throughout all the Unions of England.

The present position of our knowledge regarding vaccination is based upon evidence which demonstrates,—(1.) The *protective influence of vaccination*; and (2.) The causes which have combined to *impair its protective power*. Of these in their order.

I. *Of the Protection conferred by Vaccination—its Nature, and the Evidence of its Existence.*

The main features of the reports and works already mentioned amply illustrate how small-pox diminishes in its mortality *in proportion as efficient measures are adopted to insure perfect vaccination*. To demonstrate this statement, the progress of vaccination in Great Britain and in Germany has been compared as to its influence on mortality generally; and more particularly, it has been shown,

by comparing the statistics of vaccination from various German States with similar statistics from different districts in Great Britain and Ireland, that where vaccination is most perfectly carried out, small-pox is least mortal. The following are the general results which the Committee of the Epidemiological Society arrived at:

1. To prove the influence of vaccination in England, it is shown that out of every 1000 deaths in the half-century from 1750 to 1800 there were **96** deaths from small-pox; and out of every 1000 deaths in the half-century from 1800 to 1850 there were only **35** deaths from small-pox.

2. To prove the influence of vaccination on the Continent, it is shown that in various German States sufficient evidence can be obtained to show that out of every 1000 deaths *before vaccination was used*, **66.5** were deaths from small-pox; but that out of every 1000 deaths *after vaccination* came into use, the deaths from small-pox were only **7.26**.

3. To prove that in countries where vaccination is most perfectly carried out small-pox is least mortal, it is shown that—

(a.) In this country, where vaccination has been voluntary, and frequently neglected, the deaths from all causes being 1000, the deaths from small-pox were as follows:

London,	16	Edinburgh,	19.4
Birmingham,	16.6	Glasgow,*	36
Leeds,	17.5	Galway,*	35
England and Wales,	21.9	Limerick,*	41
Perth,	25	Dublin,	25.6
Paisley,	18	Connaught,*	60
All Ireland,		49	

(b.) In other countries, where vaccination has been more or less compulsory, the deaths from all causes being 1000, the deaths from small-pox were as follows:

Westphalia,	6	Bohemia,	2
Saxony,	8.38	Lombardy,	2
Rhenish Provinces,	8.7	Venice,	2.2
Pomerania,	5.25	Sweden,	2.7
Lower Austria,	6	Bavaria,	4

Evidence corroborative of these results has been adduced by Dr. Balfour from the records of the Army and Navy Medical Departments, where every soldier or sailor is protected by vaccination, if he has not previously suffered from *cow-pox* or *small-pox*.

1. For twenty years, namely, from 1817 to 1837 inclusive, it is shown that in Dragoon Regiments and Guards, with an aggregate

* With regard to the high rate of small-pox mortality in the towns marked by the asterisks, it was clearly shown by Dr. Stark, in Edinburgh, and by Dr. J. C. Steele (the present Medical Superintendent of Guy's Hospital in London), that such mortality was due to the neglect of vaccination. Dr. Steele, then resident in Glasgow Infirmary, called attention to the great increase of small-pox in Glasgow, as mainly coming from the Highland and Irish population, among whom vaccination was rare. Dr. Stark showed that more than eighty per cent of all the deaths from small-pox happened in children under *five* years of age.

strength during that period of 44,611 men, and a total mortality of 627, only *three* deaths were from small-pox.

2. Among the troops at Gibraltar, the aggregate strength being 44,611 men during that period, and a total mortality of 1291, only *one* death from small-pox occurred.

3. In the West Indies several epidemics of small-pox prevailed during the period, but there was *no deaths* either among the British or white troops, of whom the aggregate strength was 86,661, and with a total mortality of 6803. Among the black troops on the same station, with an aggregate strength of 40,934, and a mortality of 1645, there was not *one* case of small-pox.

4. At Bermuda, Nova Scotia, New Brunswick, Cape of Good Hope, and the Mauritius, not a death from small-pox occurred during the twenty years mentioned; and the white troops of Western Africa wholly escaped this disease, while the black unprotected population were dying by hundreds.

5. In Malta, from 1818 to 1838 inclusive, the aggregate strength of the British troops being 40,826 during these twenty years, and the total mortality 665, only *two* deaths were from small-pox. This is the more remarkable, inasmuch as small-pox raged as an epidemic all over the island in 1830, and again in 1838, destroying 1169 persons. In 1830 there died of small-pox 1048, out of a total mortality of 3407; and in 1838 there were 121 deaths from small-pox, out of a total mortality of 2583. The disease was first introduced by His Majesty's ship "Asia." The mortality among those "*not vaccinated*" was 1 in 4.7; among those "*supposed to have been vaccinated*" it was 1 in 23.4; and, lastly, among those attacked a second time by small-pox the mortality was 1 in 10.8. The native population of Malta in 1830 was estimated at 100,839 persons, amongst whom it appears that 1 in every 12.1 persons was attacked with the disease, and 1 in every 85 persons died. Amongst the military, including wives and children, the proportion attacked was 1 in 188, and the mortality only 1 in 682.

6. The most important observations are those made at the Royal Military Asylum, in which the prevalence of variola among a *vaccinated* and *unvaccinated* population, at the same ages and in exactly similar circumstances, has been traced out by Dr. Balfour (see *Med.-Chir. Trans.* for 1852).

One very obviously beneficial result of vaccination has not been so much appreciated and noticed as it ought to be—namely, that while the *epidemic influence* of small-pox greatly *increased* during the practice of *inoculation*, it has greatly *diminished* since vaccination has been adopted. Dr. Hebra, of Vienna, alludes to the fact, and incidentally remarks, "That epidemics of small-pox have been more rare, and are less malignant, since the introduction of vaccination." Definite data are to be gathered from the various reports already mentioned, which fully bear out the belief. The results may be stated as follows:

1. During ninety-one years previous to *inoculation*, there are on record 65 distinct and well-marked epidemics; which is equal to a ratio of **71.4** epidemics in 100 years.

2. During sixty-three years in which *vaccination* was practised, and that to a very great extent, there was 53 distinct and well-marked epidemics; which is equal to a ratio of **84** epidemics in 100 years.

3. During the fifty-five years since *vaccination* has been mainly practised, there have been 12 distinct and well-marked epidemics of small-pox; which is equal to a ratio of **24** epidemics in 100 years.

This kind of testimony is greatly enhanced by the fact, that epidemics never occur in the army or navy of our own country, nor in those countries where the soldiers and seamen are efficiently protected by vaccination. The details given with reference to the two Malta epidemics in 1830 and 1838 afford a striking proof of the protective power of vaccination when tested by epidemic influence; and there are records of the Danish army and navy having altogether escaped during several epidemics of small-pox in Denmark.

There is still another way in which the protective power of vaccination makes itself manifest—namely, by *the mildness of the disease in the vaccinated compared with the unvaccinated, and the almost absolute security against death from small-pox which PERFECT vaccination confers*. With few exceptions, this appears to be the universal belief of the medical profession. At various times the opinions of large numbers of medical men have been specially asked for and obtained on this point. Three distinct and very comprehensive “*polls*” may be referred to, namely,—(1.) That by the College of Physicians in London, eight years after vaccination had been adopted. (2.) An almost national “*poll*” taken by the Epidemiological Society of London about the years 1852 and 1853. The written opinions of nearly two thousand medical men in this kingdom, as well as Bombay, Bengal, the Mauritius, the West Indies, and various other places, were here expressed; and they concurred in confirming the belief in the protecting and modifying influence of vaccination in small-pox. (3.) A very extensive “*poll*,” of which a list is published by Mr. Simon, comprehends not only members of the medical profession generally, but also the members of the Medical Department of the Army and Navy, together with the opinions of foreign governments.

These “*polls*” may be held as completely decisive of the question, really practically decided in the affirmative fifty years ago. From such evidence the inference is so inevitable, “that he who disputes it is equally unreasonable as he who opposes in like manner any proposition in Euclid” (ALISON).

The actual extent of the security against death from small-pox enjoyed by vaccinated compared with unvaccinated persons, has been calculated by Mr. Simon from various sources; and it appears that the death-rate from small-pox amongst the vaccinated varies from an inappreciably small mortality to $12\frac{1}{2}$ per cent.; that amongst the unprotected the death-rate from small-pox ranges from $14\frac{1}{2}$ to $53\frac{1}{2}$ per cent.

The average percentage mortality from small-pox, stated by Mr. MARSH to occur amongst the vaccinated, is 5.24; but when vaccination is known to have been perfectly performed, as shown by the

cicatrices, the mortality is uniformly found to be reduced to less than half of one per cent.

Another very important and most interesting phase in which vaccination may be viewed as exercising a protecting influence *indirectly* over the health of the community, may be studied in those statistics which show that the *general death-rates from other diseases* have diminished, and more especially as regards *scrofulous diseases*, since vaccination became more universal. To Dr. Greenhow and to Dr. Farr in this country, and to the Statists of Sweden, we are indebted for any accurate knowledge existing on this subject. Not only has the grand total of the death-rates been diminished, but the death-rates of two special classes of diseases have diminished in a remarkable degree. These are,—(1.) Those of the tubercular order of the constitutional diseases—namely, scrofulous affections, including phthisis or pulmonary tuberculosis; and (2.) The continued fevers, especially typhoid fever—diseases belonging to the miasmatic order of the Zymotic class.

Another kind of historical evidence bearing out the protective influence of vaccination is to be seen by comparing the advertisements of old newspapers, especially those during the last portion of the seventeenth and early portion of the eighteenth century, with similar advertisements of the present day. At the former period the ravages of small-pox upon the population were beyond conception, testifying to the wide-spread epidemic influence. The description of every man and woman, as exhibited in "the villanous portraits of the Hue and Cry," showed them to have been more or less marked with small-pox, or "speckled with *pock-holes*." Nowadays it is the exception, and not the rule, to see such pits and scars amongst the population (*Quarterly Review*, July, 1855).

It is thus clearly demonstrated how vaccination has thrown the *axis* of protection over the world; and how ample, how great, and how efficient that protection may be. It has been shown to diminish mortality generally, and the mortality from small-pox in particular, both in civil and in military life, at home and abroad, and just in proportion as it is *efficiently* performed. It has been shown to diminish the epidemic influence; it has been shown to preserve the good looks of the people; it has been shown that it tends to render small-pox a mild disease compared with the same disease in the unprotected; it confers an almost absolute security against death from small-pox; and lastly, it has been shown to exercise a protecting influence over the health of the community generally. On the other hand, it is no less amply proven that "wheresoever vaccination falls into neglect, small-pox tends to become again the same frightful pestilence it was in the days before Jenner's discovery; that wheresoever vaccination is universally and properly performed, small-pox tends to be of as little effect as any extinct epidemic of the middle ages" (SIMON). Moreover, it has been clearly shown by the *systematic inspections* instituted by Her Majesty's Privy Council under the direction of Mr. Simon, that it is hopeless to expect to be free from fatal epidemics of small-pox, of greater or less extent, so long as unvaccinated children are allowed to accumulate as they have been found

to do. There is therefore the greatest necessity for vigilance on the part of every intelligent member of the community to prevent any re-accumulation of unvaccinated persons.

Four conditions are absolutely necessary to be efficiently carried out before we can hope to see small-pox eradicated through vaccination, and the aim of Jenner accomplished. These are,—(1.) The vaccination of every child must be made compulsory within a certain time after birth. (2.) *Systematic inspections* of two kinds must be constantly and periodically made by competent persons—namely, one to ascertain as to the effectual performance of the operation, as evinced by the kind of cicatrix visible. This inspection may be most conveniently made in public and private schools. Another inspection should have for its object to ascertain the numbers vaccinated within a certain territory compared with the numbers born in the same place. (3.) Every attempt at *variolous inoculation* ought to be made a penal offence. (4.) Every case of small-pox ought to be treated in strict seclusion, and be as completely as possible isolated, following out all the directions given with reference to the management of epidemics at page 228, as are applicable to the case. A quarantine regulation to enforce segregation of the sick from small-pox is of far more importance in this country than for yellow fever, which does not find a *habitat* with us. We come now to consider—

II. *How the Protective Influence of Vaccination has been Impaired.*—Since vaccination has been generally practised it has now and then seemed apparent that “*the protective power of vaccination becomes gradually weaker, and at length dies out in the individual.*” The works and reports which have been mentioned seem to demonstrate the truth of this statement; but in justice to Dr. Seaton (one of the greatest authorities on the subject) it must be stated that he does not subscribe to this belief. Indeed, in his last Report (*Appendix to Public Health Report* for 1861, p. 64), he states that where uniform care in the selection of lymph, and in the performance of the operation, was practised, the results did not favor the hypothesis that there had been any necessary deterioration of the lymph. He has seen several cicatrices, the results of the vaccinations of Dr. Jenner and Dr. Walker; but the work of the vaccinators to whom he refers (and mentions as having bestowed great care in the selection of their lymph and in the performance of the operation) will bear comparison with the results obtained by Dr. Jenner and Dr. Walker.

In 1809 Mr. Brown, of Musselburgh, near Edinburgh, published the opinion that the prophylactic virtue of cow-pox diminished as the time from vaccination increased. In 1818 and 1819 small-pox prevailed in Scotland as an epidemic, and many vaccinated persons passed through a mild form of variola. The terms “modified small-pox” and “varioid disease” about this time came into general use; and two classic monographs on the subject made their appearance, one by Dr. Monro, in 1818, and another by Dr. John Thomson, of Edinburgh, in 1820. Dr. Copland also writes that he saw and described, as early as 1823, small-pox as it affected members of the same family at different periods after vaccination, and in young persons who had been vaccinated only ten or eleven years. Contrasting such

cases, he found that the severity and fully developed condition of small-pox was generally in proportion to the length of time which had elapsed from vaccination (*Dictionary of Practical Medicine*, Art. "Small-pox," p. 815).

Again, from the evidence contained in the bills of mortality of 1825—from the experience of epidemics of small-pox in France and Italy, in 1826, 1827, and 1829—from the experience of the epidemics of small-pox in Ceylon in 1833 and 1834—and from the admissions into the London Small-pox Hospital in 1838, it has been rendered obvious that the susceptibility to small-pox, which in vaccinated persons is destroyed for some years, returns with advancing age, and becomes greater as life advances.

Some of the phenomena, also, which the practice of vaccination itself has made known to us tend to establish the doctrine of a gradual impairment of vaccine protection, due to *lapse of time*, and as a result of *physiological changes in the healthy body*. This is especially indicated by the fact, that in proportion (undetermined) to the distance of time that has elapsed from the first implanting of the vaccine virus, so is the better development of the vaccine vesicle produced by re-vaccination. It has been shown, however, from a careful analysis of cases, that the lesser protectedness of certain vaccinated persons bears at least some proportion to the number of years which had elapsed since vaccination. Any *uniform* rate of increased susceptibility to small-pox from year to year from the period of vaccination has not been demonstrated; but an increasing susceptibility to small-pox continues up to about thirty years of age at least, after which period of life it seems that the liability to contract small-pox continues to decline (HELM, MOHL, RETZIUS, MARSON, SIMON).

Dr. Balfour, of the Army Medical Department, adverted some time ago to these important facts; and there can be no doubt of the practical result to which they point—namely, re-vaccination, as a most necessary supplemental measure to vaccination. A large reduction in mortality, and in the occurrence of small-pox, can be shown to have taken place from the practice of re-vaccination, so as to leave no doubt of its practical efficacy. The records of the earliest experience of its usefulness date from Wirtemberg, 1829 to 1836. In 1833 between 40,000 and 50,000 adults were *re-vaccinated* in the Prussian army, and in about 33 per cent. of the entire number this re-vaccination "took" with perfect success. Amongst Russian soldiers at Kasan, the rate of perfect success was about 18 per cent. In the army of Denmark, from 1843 to 1847, nearly 20,000 re-vaccinations were practised, of which more than a half were attended with *perfect* success, and more than a quarter with *modified* success. Since 1843 re-vaccination has been compulsory in the Bavarian army. From that date till 1857 not even a single case of unmodified small-pox has occurred, nor a single death from small-pox. Similar good results have followed the institution of re-vaccination in the Danish army, the army of Sweden, of Baden, and in the British army also, according to Dr. Balfour's interesting report for 1859. So great, indeed, is the practical importance of re-vaccination, that in the British army a departmental order was

issued by circular, of date 21st September, 1858, and is at present in force, which ordains that "every recruit, without exception, on joining the Head-Quarters or Depot of the Corps or Regiment to which he belongs, shall be vaccinated, even if he should be found to have marks of small-pox or of previous vaccination, and that a monthly return of the results (as to (1) a perfect vaccine pustule following the operation, or (2) a modified one, or (3) a failure) shall be forwarded to the Director-General" (*Statistical Report for 1859*, p. 21).

On the other hand, it must be remembered, as Mr. Marson clearly shows, that "probably *re-vaccination* does not afford the same amount of protection that the first vaccination *well performed* does. The great object to aim at is to vaccinate *well* in infancy. This should be looked upon as the sheet anchor; and therefore a careless vaccination should be deprecated at all times, practised under the belief that, if it fails to take effect properly, it will be of no consequence, as the operation can be repeated. By such a proceeding the vaccination often takes effect *badly*, and will never afterwards take effect *properly*, and the individual may take small-pox *severely*."

It has been alleged (but sufficient proof has not yet been adduced to show) that the vaccine virus becomes deteriorated by its passage through numerous human bodies. In other words, it has been supposed that its protective influence is weakened by length of time or of use, in consequence of the long succession of subjects through whom it has been transmitted since its direct inoculation from the cow. This doctrine is opposed to the obvious pathological fact, that the specific virus of cow-pox, small-pox, and other similar diseases, multiplies and reproduces itself in the system of those who suffer in the natural course of these diseases. Considerable differences of theoretical opinion prevail upon the point. In the report of the National Vaccine Establishment for 1854 it is stated "that the vaccine lymph does not lose any of its prophylactic power by a continued transit through successive subjects." Such an unqualified belief is not, however, by any means universal, as shown in various parts of the evidence collected by Mr. Simon. It is certain that the vaccine lymph, when taken direct from the cow, seems to show an amount of infective power which is not usual in lymph of long descent; but how much of this effect is due to irritation simply, and how much to specific action, does not seem certain. Lymph direct from the cow "takes" (as the phrase is) in persons with whom lymph of long descent has failed. This is more often obvious in re-vaccinations. Lymph direct from the cow excites local changes of an intenser kind, so active, indeed, as to render caution necessary in its selection and use. The vesicle produced by it runs a full course, compared with which the progress of vaccine vesicles from lymph of long descent seems unduly rapid, and their termination premature. Also, the lymph direct from the cow renders more certain, and apparently more characteristic, the slight febrile disturbance which is proper to the action of cow-pox on the human system. This febrile disturbance is undoubtedly an essential pathological phenomenon, demanded alike for the due protection of the vaccinated

person and for the perfect development and local multiplication of laudable and efficient lymph at the spot where the specific vaccine virus was originally implanted. The more distinctly and typically the febrile action is expressed which follows the implanting of the vaccine virus, the more certainly is the person protected, and the more efficient is the local development of the lymph which has been multiplied at the site of inoculation. The development of any other febrile state, such as from cold, or other disease, is apt to hinder the development and progress of the vaccine vesicle altogether. Referring to the records of re-vaccination in the Prussian army, an extremely interesting fact is brought out by Mr. Simon, tending to confirm the doctrine that, by transmission through a succession of persons, the vaccine virus has degenerated—namely, "*that the re-vaccination of 1836, as tested by eventual resusceptibility to cow-pox were not half so stable as the vaccinations of 1813.*"

On the whole, therefore, there appears to be still room to believe that any diminution of protective influence from vaccination may be due to personal carelessness—first in the *selection* of lymph for use, as well direct from the cow as of lymph of long descent; and secondly, in the choice of cases to continue the vaccinations from—cases, for example, being chosen where the lymph of the local vesicle at the site of vaccination has been developed in the absence of the constitutional specific febrile phenomena; and in which the lymph contents of the vesicles are not only impotent, but the anatomical development and structure of the vesicles in respect of its dissepiments are at the same time incomplete and imperfect. The cicatrix, scar, or mark left by imperfect vaccination is also an imperfect cicatrix, and is capable of recognition as such. To an almost incalculable extent, the *protective power* of vaccination has been impaired by imperfect vaccination, as shown by Mr. Marson—a fact which does not seem to be duly appreciated as yet, either by the Medical Profession or by the Public.

[The results of spurious vaccination, as shown by large observations by medical men both in the United States and Confederate armies, during the late war, have been thus summarized:

(1.) There were three kinds of spurious vaccination prevalent in the American armies; (*a*) that which occurred in consequence of the loss of the specific property in the once good lymph or crust used; (*b*) that which resulted from the impairment or destruction of the vesicle as soon as it began to form, in the marching and excessive exercise of the soldier, and that resulting from the use of the sero-purulent matter of such destroyed vesicle in vaccinating other persons; (*c*) that resulting from the employment of matter from pustules or crusts that never had the genuine qualities of vaccine virus.

(2.) Scurvy and all the asthenic dyscrasie of army life, not only prevented or greatly impaired the normal operation and effect of genuine vaccinia when soldiers with such conditions were subjected to vaccination, but they frequently became the causes of certain morbid phenomena, as obstinate ulcers, &c., which caused the greater part of the evils from vaccination during the war.

(3.) In the armies it was never proved that the normal vaccine vesicle communicated any other than normal innocuous virus; but from careless-

ness in taking lymph at its perfection, and by neglecting to observe the rule never to use lymph or crusts not perfect in all respects, and free from blood or pus, frequent instances of inoculation with purulent matter or unhealthy blood happened.

(4.) By the use of matter, fluid or concrete (purulent or morbid in either case), taken from sores of any specific and enthetic character, as (a) erysipelatous and ecchymatous; (b) that of zymotic ulceration and destruction of tissues, and possessing the properties of a morbid poison; (c) syphilis, primary or secondary, whether communicated by a lancet, or contaminated vaccine points or crusts; or, as occasionally happened, the manifestation of syphilitic phenomena in connection with, or supervening upon, genuine or spurious vaccination.

(5.) The deterioration of genuine virus, by transmission through scorbutic and unhealthy persons, or where at the time of re-vaccination the protective power of a former vaccination was partially retained, or to the continued use of virus from adult soldiers many of whom were suffering from unhealthy influences, instead of using lymph from the primary vesicles of healthy infants.

(6.) The destruction or deterioration of originally good virus by heat and humidity.

These results substantiate the observations of Jenner as to the necessity of guarding against deterioration of the virus of cow-pox, which losing its specific property, ceases to be prophylactic. Army experience would go to prove that genuine vaccination is an absolute safeguard against small-pox (DR. ELISHA HARRIS, *Contributions relating to the Causation and Prevention of Disease, &c. Published by the United States Sanitary Commission, 1867*).]

The Operation of Vaccination ought to be performed in childhood, and it is ordained by law in this country to be performed within *three*, or in case of orphanage within *four months* of birth. The infant ought to be, at least, from *four to six weeks* old, before a disease, sometimes attended with considerable febrile disturbance, is ingrafted upon the constitution. Under *six weeks* of age, infants should never be vaccinated, unless in cases of urgent necessity, such as small-pox being in the vicinity. The age of *three months* is on the whole to be preferred. The child ought to be in good health, free from any eruptive cutaneous disease, and free from disorders of teething, of the bowels, or other diseases peculiar to the age of childhood, otherwise the protective influence of the vaccination cannot be depended on.

Difference of opinion exists as to the number of vesicles it is proper to graft upon the arm, and the size of them. Some believe the person to be as thoroughly protected by a small vesicle, "the tenth of an inch in diameter, as if the arm were covered with inoculated points" (CAZENAVE, ANDREW ANDERSON); and many vaccinators regard the multiplication of vesicles only as a safeguard against failure, and attach value to *one* successful insertion only of the vaccine lymph (Buchanan, *Appendix to Fourth Report on Public Health*, for 1861, p. 111). On the other hand, the official instructions issued to vaccinators in England contain the following directions: "In all ordinary vaccinations, vaccinate by *four or five separate punctures*, so as to produce *four or five separate good-sized* vesi-

cles; or if you vaccinate otherwise than by separate punctures" (for some vaccinators prefer to make long scratches, side by side or intersectingly, instead of punctures), "take special care to secure the production of *four or five separate good-sized vesicles*." This is considered necessary for securing to those who are vaccinated the full amount of protection which good vaccination confers. The superior value of several vesicles is especially insisted upon by Marson, Seaton, and Simon. They have shown a constant relation to subsist between the *number* of the sufficient cicatrices and the degree of protection afforded.

The skin covering the insertion of the deltoid muscle is the place generally chosen for implanting the specific lymph of variolæ vaccinae. The methods of operating are as follows:

1. The part of the arm to be operated upon should be grasped with the left hand, and the thumb of that hand should draw the skin with sufficient tightness, so as to facilitate the introduction of the point of the lancet with the other hand. Three or four punctures should be made near each other, for each intended vesicle. These punctures should penetrate the cuticle to the extent of a few lines in an oblique direction, so as to make a minute valvular aperture and so as to impinge upon or penetrate the *cutis vera*. The lancet used to make the puncture should be charged with the vaccine virus. It should be allowed to remain in the punctures for *several seconds*, and, in the course of its removal, the site of puncture should be compressed for a moment or so, to prevent bleeding, and also to retain the virus from the lancet's point. In the case of several punctures, it is advisable to use "*points*" of ivory or quill, or of the teeth of a comb, charged with the virus. These should, on the withdrawal of the lancet, be inserted into the punctures, and allowed to remain for several seconds, to be removed in the same way as that in which the lancet charged with the virus is removed.

2. Another mode of operation is often chosen—namely, to make an immense number of minute scratches over a very limited area of skin, and as close together as possible. In this way the number of groups of scratches will correspond to the number of vesicles intended to be ingrafted. The scratches may be made with the point of a clean lancet, and may be either parallel to each other or crossed in two or any number of directions. The number of these groups of scratches will vary according as three, four, five, or more vesicles are considered necessary, and the length of the individual scratches will determine the size of the resulting vesicle, and, to some degree, the soreness of the arm. It is necessary to remember these facts in dealing with young and delicate children, so as not to give rise to unnecessary suffering, torment, and danger. The scratches should be so slight as barely to result in the faintest possible exudation of blood, and that only after the lapse of a second or two. To the group of scratches from which blood first exudes, the "*point*" or lancet is to be applied, charged with the specific virus. The lymph containing this virus will be at once absorbed; and the blood with which the lymph is mixed should be smeared over and pressed into the other scratches in succession several times.

3. Simple abrasion of the cuticle is sometimes resorted to with very good success—namely, by scraping off the cuticle with the lancet, used as an eraser is used to remove blots from paper (*Fourth Report on Public Health*, p. 107).

Signs of Successful Vaccination.—By the end of the *second* day small spots appear elevated over the sites of the punctures, or over the groups of scratches or abrasions; and these, when examined by a simple lens, are seen to be vesicular, and surrounded by a slight redness. This stage continues for three to four days from the date of ingrafting the virus. About the *third*, but rather towards the *fourth* day, the elevation is more perceptible and more red; and by the *fifth* or *sixth* day a distinct vesicle is obvious upon it, of a whitish color, having a round or oval form, an elevated edge, and a depressed centre. Late on the *seventh*, or early on the *eighth* day, an inflamed ring or areola begins to form round the base of the vesicle, and with it continues to increase during the two following days. This areola is of a circular form, and its diameter extends from one to three inches. *On the eighth day the vesicle appears distended with a clear lymph. This is the day of its greatest perfection, and it is the proper period for obtaining the specific virus for continuing vaccination on others. The vesicle is now circular and pearl-colored; its margin is turgid, firm, shining, and wheel-shaped.*

Having reached its height on the *ninth* or *tenth* day, the development of the bright-red areola is accompanied with considerable tumefaction of the skin, with hardness and swelling of the subjacent areolar tissue. This erythematous ring is often the seat of small vesicles. By the *tenth* day, also, the febrile symptoms of constitutional disturbance are well expressed, the lymphatics of the arm are engorged, and sometimes a roseolous rash supervenes over the body. On the *tenth* or *eleventh* day the areola begins to subside, leaving, as it fades, two or three concentric circles of redness. The vesicle now begins to dry in the centre, and acquires there a brownish color. The lymph which remains becomes opaque and gradually concretes, desiccation commences, and tumefaction subsides, so that by the *fourteenth* or *fifteenth* day the vesicle is converted into a hard round scab of a reddish-brown color. This scab contracts, dries, and blackens, and about the *twenty-first* to the *twenty-fifth* day from the date of vaccination may fall off. It leaves a cicatrix which commonly is permanent in after-life. Indeed, the mark of a good cicatrix is indelible if it is not injured (GREGORY, MARSON, CEELY, CAZENAVE, SIMON).

While these local changes are in active progress, febrile phenomena become established—first, so slightly from the *fifth* to the *seventh* day, that often the fact passes unobserved; and again, more considerably during those days when the areola is about its height. The patient is then restless and hot, with more or less disturbance of stomach and bowels. About the same time, especially if the weather be hot, children of full habit not unfrequently show on the extremities, and less copiously on the trunk, a lichenous, roseolar, or vesicular eruption, which commonly continues for about a week. When vaccination is performed on such adults or

adolescents as have not previously been vaccinated, and likewise when lymph is employed which has recently been derived from the cow, the resulting phenomena, as compared with the preceding description, are somewhat retarded in their course, and the areola is apt to be much more diffuse. There is also more feverishness, and eruption is less frequently seen (Simon, *Health Report*, 1859).

Signs of Successful Re-vaccination.—When persons who have once been efficiently vaccinated are, some years afterwards, re-vaccinated with effective lymph, there sometimes result vesicles which, as regards their course and that of the attendant areolæ, cannot be distinguished from the perfect results of primary vaccination. But far more usually the results are more or less modified by the influence of such previous vaccination. Often no true vesicles form, but merely papular elevations surrounded by areolæ; and these results, having attained their maximum on or before the fifth day, afterwards quickly decline. Or, if vesicles form, their shape is apt to vary from that of the regular vesicle, and their course to be more rapid, so that their maturity is reached on or before the sixth day, their areolæ decline on or before the eighth day, and their scabbing begins correspondingly early. In either case the areolæ tend to diffuse themselves more widely and less regularly, and with more affection of the areolar membrane, than in primary vaccination; and the local changes are accompanied by much itching, often by some irritation of the axillary glands, and in some cases on the fourth or fifth day by considerable febrile disturbance (SIMON, l. c.).

Characters of the Cicatrix after Vaccination.—It seems now to have been agreed to arrange the characters of cicatrices after vaccination into the following three classes:

1. "*Typical*," "*excellent*," "*perfect*," "*good*," or "*first-rate*" cicatrices are recognized by their circular form and pale or white appearance. They are somewhat depressed, and dotted, indented, or foveolated with minute pits or depressions over the base, supposed to indicate the number of compartments in the anatomical structure of the vesicle (referred to at pages 234 and 235). In some instances there are radiations from the centre. It has been considered that the normal diameter of a cicatrix produced by a single insertion is *one-third* of an inch; that scars of larger measurement are generally of double or multiple origin.

2. "*Fair*," "*passable*," "*modified*" cicatrices possess the characters of the typical cicatrix, but they are less perfectly expressed, the contour being less regular, and the size just *within* the average above mentioned. To irregularity of contour, however, it must be remembered that scars resulting from single insertions (as in the ordinary method of puncture) are notably uniform, so that irregularity of contour, when associated with a single puncture for vaccination, indicates that the progress of the vesicle has been irregular; but where the scar results from several contiguous insertions or scratches, no such inference can be made.

3. "*Bad*" cicatrices, which must be held as denoting "*failures*," are such scars as cannot be recognized as the product of vaccination, by any circumstance beyond being found near the usual site of the

operation. Scars also having a less diameter than a *quarter* of an inch ought to find a place amongst this class; and generally, all ill-defined, faint, scarcely discernible white patches, especially such as consist of large, flat, ill-defined shiny marks. Fruitless attempts at vaccination may be also recognized by the permanent traces left of the parallel or transverse scratches employed at the operation.

It is, however, very difficult to describe the extent of differences between the results produced by different vaccinators. A large amount of *bad*, and a still larger amount of *second-rate* vaccination has been found to prevail in many districts, as the result of the inspections instituted by Mr. Simon in 1860 and 1861 abundantly testify. Medical men are found to vary exceedingly in their estimate of a satisfactory vaccine vesicle and cicatrix, or the reverse, for the standard is comparative rather than absolute (SEATON, SANDERSON, BUCHANAN). This is exactly what might have been expected, seeing that medical students are left to pick up their knowledge of vaccination where they can. In fact, practical medical education at our schools of medicine has hitherto, or until very recently, been entirely *nil* in regard to this most important subject; and no test of knowledge has ever been applied. Many men, whose estimate of the quality of the resulting cicatrices is of a low standard, can scarcely appreciate the typical character of marks which are the ordinary results of good vaccination (SEATON). Excessively small cicatrices are apt not to be perfect, and there are great varieties in the size of cicatrices of perfect character, the results of puncture. It is therefore fairly presumed that cicatrices which thus vary cannot all have precisely the same value. The hand of different vaccinators can even be recognized by the kind of marks they leave behind them. The marks of some vaccinators are conspicuous for their excellence; the marks left by others are not so; and hence there are great differences between the vaccination of districts where different vaccinators are employed. In the schools, for instance, of large towns, Mr. Seaton informs us that "where the work of many vaccinators was seen together, it was frequently possible to fit the work to the vaccinator by the kind of cicatrix."

With regard to the means of estimating the efficiency of vaccination, it seems established that "*a distinct connection subsists between the NUMBER and the QUALITY of the cicatrices and the protection conferred by vaccination against small-pox; so that it may be confidently stated that that vaccination is the most efficient from which the most and the best cicatrices result.*" The evidence derived from the records of the Small-Pox Hospital, collected by Mr. Marson, regarding the superior value of several rather than few vesicles, appears to be conclusive on this point.

These facts have been tabulated by Mr. Simon in the following form, as the result of observations made during twenty-five years, in nearly 6000 cases of small-pox contracted after vaccination, the persons having been vaccinated in different ways as regards the *number and quality* of the cicatrices:

Cases of Small-pox Classified according to the Vaccination Marks or Cicatrices borne by each Patient respectively.	Number of DEATHS per cent. in each Class respectively.
CLASS I. Stated to have been vaccinated, but having no cicatrix,	21½
" II. Having ONE vaccine cicatrix,*	7½
" III. Having TWO vaccine cicatrices,†	4½
" IV. Having THREE vaccine cicatrices,	1½
" V. Having FOUR OR MORE vaccine cicatrices,	½
UNVACCINATED,	35½

Looking, therefore, to the characters or quality of the cicatrices, and to the number of the vesicles which have given rise to these cicatrices FOUR DEGREES of protection conferred by vaccination may be specified, and the community inspected may be arranged into the following FOUR classes:

- CLASS I. Best protected—having *more* than *two* TYPICAL marks.
 CLASS II. Sufficiently well protected—having *two* TYPICAL marks.
 CLASS III. Moderately protected—having *two* or *more* PASSABLE, or *one* TYPICAL mark.
 CLASS IV. Badly protected—having BAD marks, or having only *one* PASSABLE mark.

Selection of Lymph for Vaccination.—The lymph used for vaccination ought to be taken from the vesicle on the EIGHTH day—the *day-week* after the operation—when the lymph is yet clear and the vesicle turgid, firm, shining, pearl-colored, and translucent, and before the vascular zone has reached its full development. The lymph ought not to be taken from any but perfectly “*typical*” vesicles. Inferior, or merely passable vesicles, ought not to be used to propagate lymph. Small vesicles, exhausted vesicles, or vesicles far advanced (such as tenth or twelfth day) are to be avoided. Very early lymph appears, as a rule, to give the worst cicatrices. Thus “the careful vaccinator does not indifferently vaccinate from the arms of all infants brought back on the *eighth* day, but exercises selection among them.” The fresh lymph from the vesicle ought to be ingrafted *directly* upon the arm of the child about to be vaccinated—“arm-to-arm” vaccination, as it is called. Dry, or otherwise preserved lymph, ought only to be used when fresh lymph cannot possibly be obtained. Properly dried lymph, however, seems capable of producing quite as good results as arm-to-arm vaccination; but it demands incomparably more care than it generally receives, first in its storage and afterwards in its use. It may be dried and stored on “points” of ivory or bone, or upon small pieces

* Among cases in which the one cicatrix was *well-marked* or *typical* the death-rate was 4½. Among cases in which it was *badly* marked the death-rate was 12.

† Among cases in which the two cicatrices were *well-marked* the death-rate was 2½. Among cases in which they were *badly* marked it was 7½.

of glass glued together by the dried lymph, or on lancets. These should be well charged—i. e., coated twice, or even thrice with the lymph, and rolled up in a covering of goldbeater's skin, and still further secured from atmospheric influences by an outer case of tin-foil hermetically sealed, or in a vial carefully corked, in which they may be packed with cotton, if they require to be transmitted to any distance. Glycerine has been used with success to keep the lymph liquid.

By proper care, complete and perfect vaccination may be attained under every variety of method; but bad vaccination, as it prevails at present, is almost always directly dependent on the careless employment of improperly preserved *dry* lymph, and indirectly associated with irregularity of inspection, in consequence of which the vaccinator remains unaware of the number and extent of his *failures*, and loses all the advantages of experience. "The use of the capillary tubes of Dr. Husband affords considerable advantages to the public vaccinator, especially if his district be rural—*Firstly*, Because it furnishes him with an efficient means of maintaining his supply without having recourse to extraneous sources, and thus enables him to dispense altogether with the use of 'points,' 'glasses,' &c.; *Secondly*, Because in thinly populated neighborhoods, in which experience shows that it is impossible to assemble all the children at any particular station, it enables him with equal advantage to vaccinate from house to house" (Sanderson in *Public Health Report*, 1861). For a detailed account of Dr. Husband's method of preserving lymph, the reader is referred to the *Second Report of the Medical Officer of the Privy Council*, 1859.

CHICKEN-POX.

LATIN, *Varicella*; FRENCH, *Varicelle*; GERMAN, *Windpocken*—Syn., *Wasserpocken*; *Varicellen*; ITALIAN, *Varicella*.

Definition.—*The disease consists of a specific eruption, in a series of crops, on the breast, back, face, and extremities, attended with fever, which runs a definite course in eight or ten days.*

Pathology.—This disease derives an importance which it does not of itself possess, in consequence of its resemblance to small-pox, with the modified form of which it is considered by some to be identical. It is for the most part peculiar to childhood and early adult age; but its epidemic influence is very inconsiderable, and its extension easily under control. That it is communicable has been proved by inoculation. The theory of the disease, therefore, is, that a specific poison, after a given period of latency, gives rise to primary fever which lasts from twenty-four to seventy-two hours, when the eruption appears, and runs a course of eight or ten days. The fever is much mitigated on the appearance of the eruption, and entirely subsides with it.

That fever precedes the eruption is a phenomenon observed so generally that no exception is to be found in the account given of

the disease by any writer excepting Heberden. The febrile affection is of a mild character, and though for a few hours it may seem severe, yet, perhaps, it never passes into a stage so severe as to have the tongue of a brown and coated appearance. The eruption has three stages,—that of pimple, of vesicle, and of incrustation; and after the fever has lasted from twenty-four to seventy-two hours, a number of red papulæ appear, which become vesicular, and perhaps in a few points pustular, on the first day. On the second day the vesicles are filled with a whitish or straw-colored lymph. On the third and fourth days they attain their greatest magnitude, when they become *acuminated*, and shortly afterwards they burst and shrivel, except those which contain purulent matter, and have much inflammation around their base. The fifth day they begin to crust, and in four or five days more the crust falls off, leaving for a time red spots on the skin, generally without, but sometimes with a "pit" or depression. The "pit" is permanent, and the cicatrix generally whiter than the original tissue, and the patient consequently is marked or scarred. The eruption is not at first universal over the body, but usually consists of a series of crops, which succeed each other at intervals of twenty-four hours, and die away in the order of their occurrence. The first crop usually appears on the breast and back, and afterwards on the face and extremities. The number of crops may be limited to two or three, while in other cases a new succession will appear every twenty-four hours for ten or twelve days.

Symptoms.—Of the varicella there are three forms, the *varicella lenticularis*, the *varicella conoides*, and the *varicella globata*. The symptoms of these varieties are similar to each other, their only differences consisting in the size and form of the vesicle—that of the *varicella globata* being the largest.

The fever which precedes this eruption is often as severe as that which precedes mild small-pox or measles; but it generally, though not constantly, remits on the appearance of the eruption, and does not return as the eruption approaches maturity. The urine, however, is usually little affected in the early stages, when it is often as limpid as in hysteria; but when the fever runs high, it assumes the usual febrile characters (PARKES).

The globate chicken-pox is known as the *swine-pox*, or, vulgarly, "*the hives*." The eruption consists of large vesicles not quite circular in form, but often a little larger than the pustules of small-pox, surrounded with a red margin, and containing a transparent fluid, which, on the second day of the eruption, resembles milk whey. On the third day they subside, shrivel, and present a yellow tint. Before the conclusion of the fourth day they are converted into thin blackish scabs, which dry, and fall off in four or five days more.

Diagnosis.—Dr. John Thomson, who carefully studied this disease during the epidemics of 1815 to 1821, concluded that it was impossible to distinguish chicken-pox from modified small-pox; and as their identity is still a matter of opinion, the following state-

ments (CRAIGIE) embrace the most important pathognomonic characters derived from the respective phenomena of both diseases:

1. Chicken-pox emits a peculiar odor, different from that of small-pox, and less decidedly partaking of the variolous fœtor.

2. Chicken-pox appears indiscriminately, and almost equally all over the person, beginning first on the trunk in general, and then appearing on the face and scalp; while small-pox appears first on the face and neck, and the pimples are more numerous on the face than on any other part.

3. Chicken-pox eruption is generally completed in the space of twenty-four hours, or solitary vesicles come out irregularly afterwards in different points; but in small-pox the eruption begins in the evening of the third, or morning of the fourth day, and proceeds regularly for the ensuing three days, until it is completely established.

4. While variolous pustules are on the first and second days of the eruption small, hard, globular, red, and painful, and communicate to the finger a sensation similar to that which would be excited by the presence of small round seeds under the cuticle; in chicken-pox, every vesicle almost has on the first day a hard red margin, but communicates to the finger a sensation like that from a rounded seed flattened by pressure.

5. On the second or third day of the eruption of chicken-pox, the individual bodies are vesicles containing serous fluid, and giving them a whitish aspect.

6. These vesicles are surrounded by little or no inflammatory redness, and do not naturally, and independent of external violence, proceed to suppuration.

7. Chicken-pox may be confidently distinguished from small-pox on the third and fourth days by the state of the vesicles, some of which, being left entire, are shrivelled and wrinkled, while others, whose ruptured tops have been closed by incrustation of their fluid, are marked by radiating furrows. None present depressions on the apices; and as they do not suppurate, they incrust and disappear sooner than variolous pustules.

8. The marks left by chicken-pox, when they do leave marks, present a peculiar conformation, being round or elliptical, and less frequently irregular than those of small-pox, and in general smooth and shining. Lastly, it is said by Luders, that while small-pox is formed in the *cutis vera* or corion, the chicken-pox eruption is formed in the tissue situate between the corion and cuticle (CRAIGIE, vol. i, p. 614).

Treatment.—It consists simply in abstinence from animal food, having recourse to a milk diet, and careful attention to the bowels. The patient is to be kept cool, by light coverings, and by making him repose on a mattress rather than on a feather bed.

MILIARY FEVER.

LATIN, FRENCH, GERMAN, AND ITALIAN, *Miliaria*.

Definition.—*A disease in which there is an eruption of innumerable minute pimples, with white summits, occurring in successive crops upon the skin of the trunk and extremities, preceded and accompanied with fever, anxietas, oppression of respiration, copious sweats of a rank, sour, fetid odor, peculiar to the disease. The base of the pimples and the skin around are red and irritable.*

Pathology.—As to the specific nature of this disease pathologists are not agreed. All physicians are not disposed to admit that in *miliaria* a peculiar specific disease exists, with a characteristic eruption and definite course, such as the variolous pustules and course of small-pox exhibit; by many it is believed that "this affection is almost invariably symptomatic." Certain it is, however, that a peculiar epidemic disease prevailed in different parts of Europe at different periods in the world's history, the nature of which is described in the definition; and although in this country it seems to have disappeared, yet there can be no doubt that a specific disease of this description prevails epidemically in many parts of continental Europe and Asia. The disease of these epidemics has been described under the various names of "sweating sickness," "miliary fever," "sudatoria," "miliara," and the like. Rayer has given the most accurate account of the disease; and I had an opportunity of witnessing a great number of cases of it amongst the Turks, in their military hospitals at Scutari, during the war against Russia in 1854-1856. The temperature and physical climate of that place, combined with the relaxed habits of the Turks, appear to be favorable to the development of such a disease. The best accounts of it are those of Borsieri and Rayer.

Symptoms.—The fever which precedes the eruption is ushered in by chills, intense and general, shivering, anxietas, oppression of the chest, restlessness, a sense of great feebleness and imminent fainting, with pains in the head, loins, and limbs. In a few hours, nausea, flushing, and profuse sweating supervene, but without any diminution of the dyspnoea, the anxietas, or pectoral oppression, but rather with an aggravation, in the form of short, irregular, panting, and sighing breathing, as if proceeding from a sense of weight under the sternum, with a feeling of internal heat, wandering pains, and sometimes cramps of the hands and calves of the legs. The pulse is generally rapid, small, and feeble; in a few cases hard; often, variable, irregular, or intermittent at every ninth, twelfth, or sixteenth beat. The tongue is coated with a white, foul, or yellow fur, indicative of a sluggish condition of the alimentary canal; and the bowels are constipated throughout the disease. The sweat which accompanies this febrile state is profuse, and emits a peculiar smell of a rank, sour, fetid odor. From the fifth or sixth day, up to the twenty-first, an itching sensation is felt in the mammary and epigastric regions, and inner surface of the arms, and the skin of those parts is found to be diffusely red, rough, and irregular, with numerous elevations not larger than pin-heads. In a short time the summits of these become pearly-white, the cuticle being elevated by a slight opaque sero-albuminous fluid,—crop after crop breaks out, and continues from three to seven days, followed by a corresponding desquamation of the cuticle. This eruption is generally confined to the neck, breast, mammary and epigastric regions, and the inner surface of the loins and legs. In severe cases, miliary vesicles appear at the junctions of the skin and mucous membranes, and there they are apt to become aphthous.

A deranged state of the gastro-enteric mucous membrane, indi-

cated by nausea and vomiting of bilious matter, acid eructations, flatulence, and diarrhœa, frequently complicate the disease. Two forms have been described,—namely, a mild and malignant. The malignant is rendered so chiefly by the occurrence of violent inflammation in some of the internal organs, especially of the stomach, lungs, kidneys, or brain; and the danger of the disease is chiefly due to these complications. Such malignant forms have been known to prove fatal in two or three days, but more frequently in from seven to twenty-one.

The Treatment of the disease appears to consist in cooling drinks, purgatives, and antiphlogistics, as prescribed by the Italian medical officers who commonly attend on the sick in Turkey.

MEASLES.

LATIN, *Morbilli*; FRENCH, *Rougeole*; GERMAN, *Masern*; ITALIAN, *Rosolia*.

Definition.—*The eruption in crops of a crimson rash, consisting of slightly elevated minute dots, disposed in irregular circular forms, or crescents; preceded by catarrhal symptoms for about four days, and accompanied with fever. It affects the system only once; and sometimes prevails as an epidemic. The eruption lasts six or seven days, and the whole duration of the disease is completed in from nine to twelve days.*

Pathology.—That a poison is absorbed in cases of measles, and infects the blood, there can be no doubt, inducing, after a period of incubation of thirteen or fourteen days, a continued fever, which does not remit on the appearance of the eruption. The fever thus established at the end of three, more generally of four, and in some few instances of five days, is followed by a certain secondary or specific inflammation of the skin and of the mucous membranes of the eyes, nose, mouth, fauces, and bronchia. In a few cases the poison has certain tertiary actions, and produces inflammation of the substance of the lungs, or of the pleura, which may greatly prolong the illness.

The pyrexia may greatly vary in intensity, but it is uniformly present. The fever which precedes the local lesions is termed the primary fever; and the premonitory phenomena of cough, sneezing, and general *malaise*, are usually more prolonged than in the other eruptive fevers. It does not always happen, however, that the functions of the mucous membrane are disordered, as well as the cutaneous surface. There are cases in which no catarrhal symptoms exist, and such cases are described as "*morbilli sine catarrho*." Such cases occur during epidemics of the disease, and are but few in number.

Since the affection of the skin is uniformly present, while that of the mucous membranes is sometimes absent, the cutaneous eruption is necessarily the great characteristic of the disease; but the morbillous eruption being evanescent after death, we can only imperfectly trace its pathology. It first appears as a circular spot or blotch, similar to a flea-bite, slightly prominent, and scarcely sensible to the touch. Its color is of a pinkish red, or deep raspberry

hue, and in rare instances, as in the *morbilli nigri*, is livid or black. In severe cases, especially if the patient be of tender age, the eruption assumes a papular form, and, when at its height, occasionally a vesicular form, the latter being most common on the arms, the neck, or the breast. The color of the eruption is evanescent on pressure, but returns on the finger being removed.

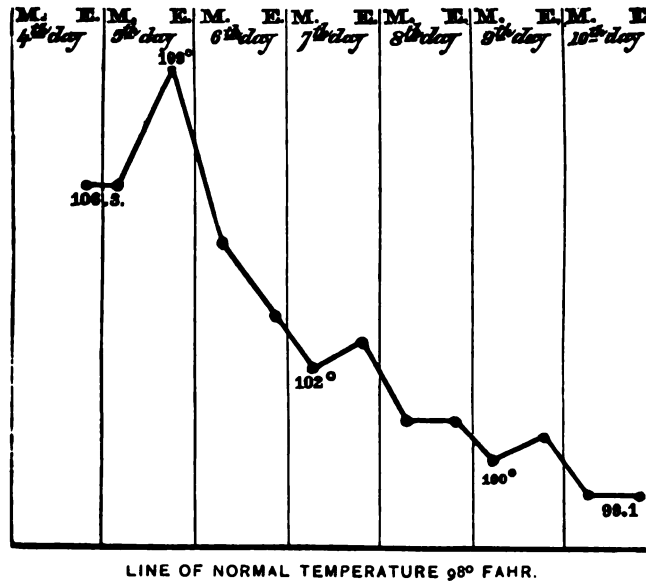
The patches of the eruption are extremely numerous, so that little of the healthy skin intervenes between them; and they not unfrequently become confluent, forming large maculæ, sometimes of a semilunar form. The principal seats of the eruption are the face, back, and loins; the parts least affected are the pudendal and popliteal regions. The inflammation of the cutaneous texture extends in some degree to the subjacent areolar tissue, for the face is tumid and swollen, but not so as to close the eyelids.

The eruption does not at once cover the whole body, but occurs in three crops, each of which follows the other at an interval of twenty-four hours, the duration of each crop being from three to four days. The course of measles, then, in its most simple uncomplicated form, is that on the third or fourth day of the primary fever, which is continuous, the first crop of the eruption appears on the face, neck, and upper extremities; on the following day the second crop covers the trunk; and on the third day the third crop appears on the lower extremities, so that the whole body is covered with the eruption, which is then at its height. On the following day (the fourth of the eruption) it begins to decline from the face, neck, and upper extremities; and on the next day it fades from the trunk. On the sixth or seventh day it is evanescent over the whole body, and terminates by resolution, followed by a furfuraceous desquamation of the cuticle generally. The maximum of the fever is reached about the fifth day, and may last from twelve to twenty-four hours; and it is immediately followed by a rapid and almost complete defervescence, the temperature sinking in one night two or more degrees (Fahr.). It continues to decrease throughout the following morning and day; and on the second day from the beginning of the defervescence the normal temperature is arrived at. It is only in very severe cases that this steady decrease is prolonged beyond twenty-four or forty-eight hours more. In severe cases the decrease of temperature may be slower and more protracted; but if the defervescence be more prolonged, it is a fair ground for suspecting some untoward complication.

A similar course of temperature, as regards defervescence, has been observed to obtain in cases of erysipelas of the face, but the fastigium lasts longer, and the epoch for the commencement of the defervescence vacillates between the fourth and eighth days.

It is the amount of fever present—*i. e.*, the increased bodily heat, as measured by the thermometer—which greatly aids in deciding diagnosis and prognosis. From this point of view slight, severe, and complicated cases are to be distinguished.

THE FOLLOWING DIAGRAM REPRESENTS THE TYPICAL RANGE OF TEMPERATURE IN A CASE OF MEASLES. THE RECORDS INDICATE MORNING (M.) AND EVENING (E.) OBSERVATIONS, COMMENCING ON THE EVENING OF THE FOURTH DAY OF THE DISEASE:



During the period of incubation, slight feverishness, depression, and catarrh are present in adults; but when the disease is expressed, its commencement may be marked by violent shivering, or merely by chilliness, characteristic of catarrhal fever. The inflammation of the mucous membrane, of the eyes, and nasal fossæ, indicated by more or less constant sneezing, generally commences either with or before the primary fever, and consequently precedes the eruption by some days. This inflammation is perhaps, for a few hours, confined to fixed spots, and is marked by itching at the mucous orifices; then it becomes diffuse, and quickly changes to the serous; for a profuse watery discharge from the eyes and nostrils shortly follows, technically termed "coryza." This affection usually continues till the decline of the eruption, and in some cases to a later period. Children, as a rule, are not anxious to seek their beds; even with a temperature of 104° Fahr. they are still able to remain up; but in cases of pneumonia, with the same temperature, they desire to lie down at once.

The temperature rises rapidly towards the breaking out of the eruption. Its rise is steady to the *fastigium*, but if remissions are marked, they occur in the morning. If the fever is high before the eruption for many days, it indicates a severe case, and is apt to be attended with such nervous derangements as are indicated by somnolency, jactitation, or delirium. The *fastigium*, or maximum of temperature, generally coincides with the period of the eruption; and simultaneously with this increase of the fever the nervous

symptoms are apt to predominate. In the majority of uncomplicated cases the temperature falls within the first *twenty-four* hours after the appearance of the red spots, and the *fastigium* in such cases does not extend beyond *twelve*, and in a few cases beyond *twenty-four* hours. Protracted *fastigia* are always connected with severe cases.

In some cases the *defervescence* is completed within *twenty-four* hours—an example of complete crisis. But pauses sometimes occur to interrupt this rapid *defervescence*—pauses which may extend from *twelve* to *thirty-six* hours. Such are examples of protracted crisis, and are always anxious cases. With regard to prognosis, it is found that high fever, having small daily fluctuations, especially with the coexistence of nervous derangements, delirium, and the like, are very unfavorable symptoms. Prolific eruptions are always more favorable than scanty exanthema. Short duration of the *fastigium*, rapid *defervescence*, and speedy disappearance of the eruption, are the most favorable events. On the other hand, exacerbations are always more or less unfavorable.

Of the complications of measles, the most important is catarrhal pneumonia; and a fatal issue is apt to ensue in cases of young children. In such cases the pulse and respirations are enormously accelerated, the face is flushed, and the movements of the body are lively. The contractions of the heart are then apt to abate in frequency, and the breathing to become very inefficient; while simultaneously with diminishing expectoration the breathing surface of the lungs becomes less and less, owing to the bronchiæ being rendered more and more impervious. Portions of lungs then collapse, and others become emphysematous.

The mucous membrane of the mouth and fauces in most of the severe cases inflame, but the inflammation differs from that of the eyes and nose in not being accompanied by any discharge. In other respects it is exactly similar to the cutaneous eruption, for a number of exanthematous patches, more or less confluent, are seen upon the palate, uvula, tonsils, and *velum pendulum palati*, and they, equally, terminate by resolution. They appear also at the same time with the eruption on the face, neck, and upper extremities, but do not decline till the eruption fades from the body generally.

The cough and expectoration, which indicate the attack and accompany it, are constant, and the latter shows that it partakes of the same serous character as that of the nasal and ocular membrane.

When the substance of the lungs is thus affected, a serous exudation pervades that tissue, and the quantity of fluid effused is frequently so considerable as to stream from the lung after death, as soon as its tissue is divided. In severe forms of the disease either the red or gray hepatization of the lung may supervene; but these results are rare. The pleura does not at all times escape the morbid action; and the diffuse, the serous, the adhesive, and even the purulent inflammation, may invade that tissue, and either destroy the patient or retard his convalescence. Few analyses of the urine in cases of measles have been made. Albumen is extremely common in some epidemics, and appears simultaneously with the eruption: it may then disappear, and reappear during the fading of the

rash. Blood in small quantities in 1854 the recovery was the greatest (P.

Symptoms.—The symptoms of the consecutive local lesions are extremely few, for no without the secondary or it is supposed sometimes to be cutis, and to exhaust itself *sine catarrho*." The varying physicians to consider the—namely, the "*morbilli miti*

The primary fever may in for a few days with symptoms latter is the case; but in no afterwards prolonged, and at of great intensity. Although severity of the local lesions, yet no instance is known of the patient being overwhelmed or destroyed by the general depressing action of the poison, as is the case in typhus fever or scarlatina. The depressing powers of the poison, however, are considerable, and are often sufficient to confine the patient to his bed for a few days, and to leave him for a short time after the disease has subsided, weak and debilitated. The type of the fever of measles consequently greatly differs from that of typhus or of scarlatina, and the formidable brown tongue, so grave a symptom in the latter, is hardly known in the former, or only seen in a few fatal cases.

Morbilli Mitiores.

The essential characters of this affection are, that the poison produces primary fever, and a specific inflammation of the skin and mucous membranes,—the defervescence of the fever taking place while the eruption fades.

The symptoms may be divided into three stages: the first embraces the primary fever, or the period before the eruption, and may last from three to five days; while the second stage embraces the period of the eruption, and lasts from six to seven days. These two stages very commonly comprise the whole disease, whose usual course is then from nine to twelve days. The third stage includes any inflammatory action which may be caused by the tertiary action of the poison, and only occasionally exists.

The early symptoms of the primary fever are seldom severe, and greatly resemble those of an ordinary acute catarrh. They are, shivering, alternated with heat, frequent pulse, headache, derangement of the bowels, sometimes accompanied by nausea and vomiting; and these affections are so considerable that the patient usually takes to bed. At the end of a few hours the fever becomes continued, and the specific action of the poison commences by the mucous membrane of the eyes and nose inflaming, so that the light is painful; the senses of smell and taste are lost, followed by a copi-

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ous discharge of serum fixed from causes not quite have this effect, or any-hereditary or acquiredThe buccal and the malignant nature of same time, and this are to be looked does not remain long late, is by hoarseness, less an rhoea, and may result, in many

symptoms are apt to predominate the nose and eyes, attended with more cases the temperature.

the appearance of the bronchial membranes may become affected at the not extend beyond patient is then troubled with a frequent cough, hours. Protruspecularity, that it occurs in paroxysms. The cough

In some it till about the seventh day, and is often accompanied hours—ness, by a sense of constriction across the chest, by diarrhoea to intend sometimes by ischuria. The duration of this first stage from be three, four, five, or even six days.

The second stage commences with the appearance of the eruption, whose course and character have been described. On the appearance of the eruption the fever is often aggravated, but the distressing nausea and vomiting seldom last beyond the fourth day. The fever, therefore, together with the coryza, sneezing, coughing, hoarseness, and diarrhoea, continue with unabated severity till the eruption has reached its height, and is fully out over the whole body, which is on the third or fourth day after its first appearance. From this period, in favorable cases, all the symptoms begin to decline; and on the eruption disappearing, the cuticle desquamates, and the disease terminates on the ninth, tenth, or eleventh day from its commencement.

In a few cases, however, on the subsiding of the eruption, or about the ninth, tenth, or eleventh day of the disease, and in some instances earlier, the pectoral symptoms do not subside as they ought to do, but the tertiary actions of the poison are set up, and inflammation of the substance of the lungs or of the pleura takes place, prolonging the duration of the disorder, and endangering the life of the patient. The inflammation of the bronchial membrane is denoted by the expectoration either of a thick viscid mucus or of pus, and which may or may not be streaked with blood; while the mucous or sonorous rattle will point out the peculiar seat and extent of the mischief. If the substance of the lungs be inflamed, the breathing is more difficult, the cough more troublesome, and the countenance livid; but the loud mucous rattle which accompanies it seldom allows us to hear crepitation, or to determine the absence of respiration in any given portion of the lung. If the pleura be inflamed, we have, in addition to the cough, severe pain in the side, and an impossibility of filling the chest with air, except in a very limited degree. This condition is often accompanied by dulness on percussion, by bronchophony or ægophony, assuring us that fluid is effused into the cavity of the chest.

Morbilli Graviores.

The main characteristic of this severe form of measles is the eruption becoming suddenly black, or of a dark purple with a mixture of yellow. The early writers on measles describe this form of the disease as being much more common in their time than we find it to be in the present day. Sydenham considers this appearance as extremely formidable, and that persons so seized are irrecoverably lost, unless they are immediately relieved by bleeding and a cooler regimen. Willan writes that he has seen this discoloration, but thinks more lightly of it.

The eruption is sometimes greatly delayed from causes not quite manifest. Excessive purging is thought to have this effect, or anything which greatly debilitates the system, hereditary or acquired unhealthiness of constitution, or the peculiarly malignant nature of the disease. The occurrence of the eruption is therefore to be looked for with anxious care, as the appearance of it, even though late, is in itself a favorable indication.

If the eruption suddenly disappears, or "goes in," it is no less an unfavorable omen, and is apt to be followed by dangerous results, diarrhœa, dyspnœa, coma, convulsions, all which unfavorable signs may again disappear on the reappearance of the eruption.

[*Camp Measles.*]

Measles is one of the most formidable of camp diseases, and prevailed epidemically to a great extent in the United States and Confederate armies during the war of the rebellion. It happened chiefly amongst the regiments just organized at the State depots, and regiments in camps and barracks soon after muster into service, and young recruits. The epidemics occurred in the fall, winter, and spring. The disease was much more common in the regiments raised or recruited in rural districts than in those from the large towns, its subjects not having been previously exposed to its contagion. The mortality rate was large. In the general field hospital at Chattanooga, it was 22.4 in 100 cases. In General Hospital, No. 1, Nashville, it was 19.6 in 100, or nearly 1 in 5 (BARTHOLOW). Many died from the sequelæ, or became permanently disabled. Contagion from a specific poison, acting upon unprotected persons, was unquestionably the means of propagation of the disease. Dr. Bartholow states: "In one regiment which came under my observation, every man contracted measles who had not had it in early life. This was the rule in all regiments exposed to the poison. This statement is supported by the fact, that in 100 cases [analyzed], 91 had not suffered at any period of life from measles, and only 9 supposed that they had had the disease, but were not at all certain" (*Contributions relating to the Causation and Prevention of Disease, and to Camp Diseases, United States Sanitary Commission, 1867*).

The symptoms, intercurrent disorders, secondary affections, and morbid anatomy, of Camp Measles differ in no essential particular from those met with in the disease amongst civilians.

The great losses to the service from the disease, both by death and discharge from its disqualifying effects, make it a matter of moment to adopt, when practicable, the means of securing armies from these results. The value of the soldier is much increased by his having gone happily through an attack. The protective measures are: (1.) Exposing to the contagion at a favorable season, and under the best hygienic conditions possible, those who may be liable from previous exemption. (2.) Isolating the cases of the disease when they occur, and preventing exposure of those obnoxious to the specific poison.]

Diagnosis.—The diseases with which measles may be confounded are scarlet fever and some forms of syphilitic eruptions. The diagnostic symptoms between measles and scarlet fever are numerous; for there are many differences, both in the general course of the fever, the ranges of temperature, and particular symptoms of these

diseases, by which they may readily be distinguished from each other. Thus, the periods of the latency of the poisons are different, that of scarlet fever being from two to ten days, while that of measles is from ten to sixteen days. The eruption in scarlet fever seldom appears later than the second day of the primary fever; in measles it is delayed till the fourth day. In scarlatina the exanthematous patches are large, and the surface they cover ample; but in measles they are not larger than flea-bites, and when most confluent the clusters are small, sometimes forming crescentic patches. The color is also different, being of a bright red in scarlet fever, while in measles it partakes more of a pinkish red or raspberry hue. The affections of the mucous membranes are also different in the two diseases. In scarlatina the tonsils are almost always greatly enlarged and ulcerated, while in measles they are little or not at all affected. In scarlatina the eyes are free from coryza, while in measles this is the most prominent symptom. The tertiary actions of the poison are also different, being, in scarlatina, inflammatory affections of the joints, and dropsy; while in measles they are inflammations of the lungs or pleura; and, lastly, in measles the fever usually subsides on the disappearance of the eruption; but in scarlatina the fever often continues many days or weeks after the eruption has run its course, or till the sore throat has healed.

Prognosis.—The mortality from measles greatly varies in different years. During each of the four years previous to 1858 the proportion of deaths from measles in every 1000 deaths from other causes has been, in 1851, 24.107; 1852, 14.599; 1853, 11.818; 1854, 21.463. Percival says, that out of 3807 cases of measles, 91 died, or 1 in 40. Watson says, that in one year, at the London Foundling Hospital, 1 in 10 died; and in another 1 in 3. In the same establishment in 1794, out of 28 cases none died; in 1793, out of 69 cases, 6 died; in 1800, out of 66, 4 died: and the aggregate of these data will give us an average of 1 death in 15; so that the prognosis in every case of measles is favorable in the first instance. The prognosis, however, is more favorable in the country than in large metropolitan towns; for it appears by the Registrar-General's reports that the proportion per cent. of the population that died of measles in London is much greater than in England and Wales.

The chief danger arises from bronchial and pulmonary inflammation, and the danger of this is greater after the disease has begun to decline than during its progress. An epidemic of measles occurred at Kiel in 1860. In the fatal cases the chief cause of death was a peculiar state of the lungs, which in part were collapsed, with *foci* of purulent infiltration in various parts, or a condition of carnification. Intense bronchial catarrh was present, extending to the minuter ramifications of the air-tubes, but not of a croupal character (Virchow's *Arch.*, vol. xxi, p. 65; *New Syden. Society Year-Book*, 1861, p. 132). In strumous patients measles may end in the development of miliary tubercles in the lungs; increasing cough, emaciation, and a harsh, dry skin being the symptoms of such an untoward result.

Croup sometimes supervenes, and cuts off young patients. It

tends to be of the asthenic type, and is not unfrequently preceded by diphtheritic inflammation of the fauces, which gradually passes down to the larynx.

Diarrhœa is another danger to be encountered. During convalescence there is a tendency to looseness of the bowels, but which, if moderate, ought not to be counteracted, as it is commonly rather advantageous; but if suffered to continue, the consequences may be fatal.

Catarrhal ophthalmia, if the constitution be strumous, must also be watched for, and, if possible, prevented.

Measles, in any of the malignant forms described, is highly dangerous; and the danger is greater in the old than in the young—in cold than in warm weather.

Causes.—Measles were first noticed at the same time and in the same country with scarlet fever, and the two diseases have subsequently followed nearly the same course. They now prevail all over the world, are little influenced by season, are believed to be constantly in existence somewhere, and occasionally epidemic.

Measles, though incidental to every period of life, are most frequently contracted in childhood, when it is difficult to trace the effects of accidental circumstances, so that our knowledge of the predisposing causes is most imperfect. Both sexes, however, appear to be equally liable to this affection. With respect to the influence of season, it is generally supposed that measles break out most readily in the beginning of winter, increase till the vernal equinox, and then tend to subside towards the summer solstice. The deaths, however, from this disease, registered in England and Wales, show that the influence of season is exceedingly trifling.

Propagation of the Disease by Direct Communication and Infection.—It is admitted by all authors that a patient laboring under measles generates a poison which may be communicated directly, or which may contaminate the atmosphere with an impalpable poison. Like scarlatina, measles is thus eminently communicable; and in like manner no susceptible person can remain in the same room, or even in the same house, with an infected person, without hazard of taking the disease. In the year 1824 it was imported into Malta by some children belonging to the 95th regiment, and spread extensively in that island, so that many natives died. This circumstance was the more remarkable, as measles had not been in the island for many years. The *inferting distance* of this poison, it will be plain from what has been stated, must be considerable; indeed, it is often very difficult to isolate the disease in public schools, or other large establishments, where it sometimes appears.

The fact of measles being communicable has often been proved; but some difference of opinion exists as to the possibility of communicating the disease by inoculation. Healthy children have been inoculated either by blood drawn from the arm of a patient suffering from measles, or with serum taken from the vesicles which are occasionally found intermixed with the eruption,—an experiment which appears to have been first made by Dr. Home, with a view of producing a mild disease; but as no such result has been obtained,

the practice has been abandoned. Many trials of this kind have failed to produce the disease, yet on the whole, successes are sufficiently numerous and varied to warrant the statement that a specific poison communicates the disease.

This disease is also propagated by *fomites*. The strictest demonstration of this fact is, that the disease has been communicated by direct application of substances impregnated with the virus in the attempts to inoculate the disease; it is also proved by the fact that children's clothes sent home in boxes from schools where the disease has raged, communicate the disease; and also by the same circumstance resulting when susceptible children have lain in the same beds, or in the same room, shortly after it has been occupied by patients suffering from the disease. Cold weather appears favorable to the development and propagation of measles. No age is exempt, from the fœtus in the womb to the second childhood of old age; but it is much more frequent in children than in adults, and there are few who have not an attack of measles at some period of life.

The morbillous poison having once produced its specific effects, as a general principle, leaves the patient exempt from all liability to a second attack. This law may be considered as proved both by Willan and Rosenstein—the former affirming that, after an attention of more than twenty years to eruptive complaints, he had not met with an individual who had twice had "febrile rubeola;" while the latter states that in a practice of forty-four years he had met with no instance of a second infection. There are, however, occasional exceptions to the rule. One variety of this disease—namely, the *rubeola sine catarrho*—is supposed to afford no protection against an attack of the *rubeola vulgaris*. There are many exceptions, however, to the non-susceptibility of persons who have passed through the *rubeola vulgaris*; for Burserius, Robedieu, Home, Baillie, Rayer, and Holland, have all seen instances of a second attack of the measles in the same individual.

The period of latency of the poison of measles is determined to vary from ten to sixteen days. It seems also ascertained that the specific poison of measles is generated as soon as the primary fever is established, and before the eruption appears.

Treatment.—The nature and course of measles differ from scarlet fever not only in the fever being much less depressing, but in running a shorter and more certain course, and in having no tendency to terminate in ulcerations or mortifications of the skin. The constitution during measles is little impaired by the short continuance of the disease, and consequently admits of a more strictly antiphlogistic treatment.

As no antidote is known to the poison of the measles, the disease must run its course. The rule, therefore, is to interfere as little as possible as long as the disease is pursuing its normal course, and merely to attempt to moderate and subdue symptoms when they threaten danger.

The *morbilli sine catarrho* is usually of such a mild form as to require no other treatment than a milk diet, the customary attention to the bowels, and the prevention of exposure to cold and wet.

Measles will not bear exposure of the surface of the body to cold so well as either scarlatina or small-pox, on account of the great tendency to bronchial and pulmonary inflammation. Children must therefore be watched night and day to prevent them lying uncovered, and special care must be taken to avoid exposure to cold during convalescence. In the *morbilli mitiores* the cough, the frequent vomiting, and the heavy catarrhal symptoms which so generally attend the primary fever, render medical attendance necessary from the first moment of the attack. The treatment of these symptoms, however, and also of the eruptive stage, as long as the patient continues free from any serious inflammatory affection of the lungs, need not necessarily be active, it being sufficient to alleviate the cough, allay the vomiting, and check the catarrh by some of the large class of saline laxatives, linseed tea, or mucilaginous mixtures, to which *antimonial wine* may be added if necessary, as a diaphoretic, and to subdue high vascular action. In making a selection from these, the physician must be principally guided by the state of the bowels and the condition of the stomach of the patient. If the bowels be constipated, the milder purging salts, as the *sulphate of magnesia*, are to be preferred. On the contrary, if the patient be purged, and the vomiting distressing, a neutral mixture or effervescing draught will be found most beneficial. There are many persons in whom the cough and catarrh are the most urgent symptoms; and in such cases, if the stomach be quiet, the *liquor ammoniæ acetatis*, in half ounce doses, combined with *camphor mixture*, from its more powerful action on the skin, is an excellent substitute. Another remedy, equally or perhaps still more useful, is *ipecacuanha*, of which from one to two grains may be given every four or six hours. Some practitioners prefer antimony to ipecacuanha, but antimony appears, at least in large doses, to act in some instances perniciously on the lungs.

The treatment which has been specified is, in most cases, all that is necessary throughout the whole course of the disease; and the greatly extended experience of Willan hardly enabled him to enlarge it. He was of opinion, however, that an emetic, given on the second or third evening, *somewhat* alleviated the violence of the catarrhal symptoms, and contributed to prevent the diarrhœa which usually succeeds measles. An emetic is especially useful if the disease be threatened with croup as a complication. During the eruption, he adds, "I have not observed any considerable effect from antimonials or other diaphoretics." Bathing the feet every evening seems a more beneficial application. Emulsions and mucilages afford but a feeble palliation of the cough and difficulty of breathing. With respect to opiates, they are not generally advisable: in the early stages especially, according to Willan, opium produces an increase of heat and restlessness, without conciliating sleep.

The catarrhal symptoms are frequently accompanied, even in the very earliest days of the disease, with much bronchial inflammation, and sometimes with pneumonia; or these affections may occur at any later period, after the decline of the eruption, from the tenth to the twelfth day of the attack. Although experience has shown

that bleeding may be practised with impunity in the very first onset of the disease, or at any subsequent stage, if the constitution of the patient is otherwise good, yet it is very rarely necessary to bleed before the subsidence of the eruption; for, if we wait that event, we "usually find the pulse become moderate, and the uneasy, laborious respiration terminate in twenty-four hours. This oppressed breathing is common to other eruptive fevers; and if it were universally to be considered as an indication for bleeding, the practice would often be more fatal than the disease" (WILLAN). If, however, pneumonia be threatened, blood should be freely but not extravagantly taken; for it should be remembered that although some children bear the loss of blood well, yet that others are long in recovering from it, even when the quantity drawn is small. In children, then, below ten years of age, when it is considered necessary to withdraw blood from them, it is more prudent to take blood frequently, and in small quantities, than in a large quantity at once. We should likewise be content with moderating the symptoms; for, as the inflammation depends on a morbid poison, it has a course to run, and does not admit of a cure. The bleeding should also be more moderate during the eruption than after it; for we have a right to look for a diminution of all the symptoms as the eruption naturally disappears. If pleurisy alone supervenes, leeches or cupping to the chest will be sufficient, without venesection. Blisters, ipecacuanha, and mercury, are amongst the best *adjuvantia* to bleeding in severe cases. Mercury is best used in the form of friction with blue ointment over the chest, a little croton oil being added to promote its absorption (ANDREW ANDERSON). In cases where miliary tubercle may be suspected to grow, good results have been obtained from quinine, nutritious diet, and wine. In the Kiel epidemic already noticed, leeching and emetics, employed moderately, were unsuccessful; but the alternate application of towels dipped in hot and cold water had very good effects, but required to be continued for hours or days. The disease was very prone to relapse, and never pursued the typical course of a simple pneumonia.

During the whole course of measles it is necessary to enjoin an abstinence from all animal food, and to limit the patient to a low diet and to slops. The chamber should be of a moderate temperature (60° Fahr.), not subject to any sudden change from heat to cold, and the strictest cleanliness should be observed. With a view to protect the eyes, the room should be kept dark, so that the patient may be prevented reading, or using his eyes. In large establishments separation is necessary, to prevent spreading of the disease, if possible. Should the eruption disappear or be retarded, and untoward symptoms appear, such symptoms must be carefully studied, as prompt measures may be demanded, with the object of bringing the eruption "out," and subduing internal irritation or inflammation. A most efficient help is the hot or vapor bath. Warm drinks may also be given; and if there are no bronchial symptoms, or evidence of cerebral oppression, a dose of compound powder of ipecacuanha will be of service, proportioned to the age of the patient.

When convulsions occur in children, hot foot baths sometimes give relief, as well as sinapisms to the limbs; after which, if they do not subside, blood must be taken by leeches from the temples; and it is in all cases necessary to determine the most probable source of the irritation, giving rise to the convulsions—*i. e.*, whether they depend upon the specific poison of the disease, upon dentition, or upon intestinal irritation or cerebral disorder. Diarrhœa should not be checked suddenly, but kept under control.

SCARLET FEVER.

LATIN, *Febris rubra*; FRENCH, *Scarlatine*; GERMAN, *Scharlachfieber*; ITALIAN, *Febbre Scarlatina*.

Definition.—*A febrile disease, the product of a specific poison, which is reproduced during the progress of the affection. On the second day of the illness, or sometimes later, a scarlet efflorescence generally appears on the fauces and pharynx, and on the face and neck, which spreads over the whole body, and commonly terminates in desquamation from the fifth to the seventh day. The fever is accompanied with an affection of the kidneys, often with severe disease of the throat, or of some internal organ, and is sometimes followed by dropsy. The disease runs a definite course, and as a rule occurs only once during life.*

Pathology and Symptoms of the Disease in its Varied Forms.—After a definite period of latency, the peculiar poison of scarlet fever induces a disorder of the blood, which is, in the first instance, made manifest by a febrile state and a disturbed condition of the great nervous centres. The primary fever having lasted for one, two, or three days, does not entirely subside, but the secondary actions of the poison are set up as a peculiar eruption, preceded, followed, or accompanied by a sore throat. The eruption runs a course of from six to eight days, but the duration of the affection of the throat is more indefinite, and varies from eight to twenty, or more days. The fever continues during the eruption, and as long as the sore throat exists; but this being terminated, it subsides, and the disease is ended. In a few instances, however, tertiary results succeed, as dropsy or inflammation of the joints, diseases quite as formidable as any which had preceded them. As in ordinary fever, the poison of scarlet fever acts on the brain and its membranes, often causing the usual forms of inflammation of those parts, modified in their course and effects by the nature of the specific febrile disease.

That fever precedes the specific actions of the skin in this disease is so general a rule that it has few exceptions; and the pyrexia has been occasionally so severe as to destroy the patient before the more specific lesions of the disease have been set up. Dr. Andrew Anderson writes that he has seen death take place in six hours from the commencement of the disease—the child, in fact, dying poisoned (*On Fever*, p. 77). In suddenness of danger it thus ap-

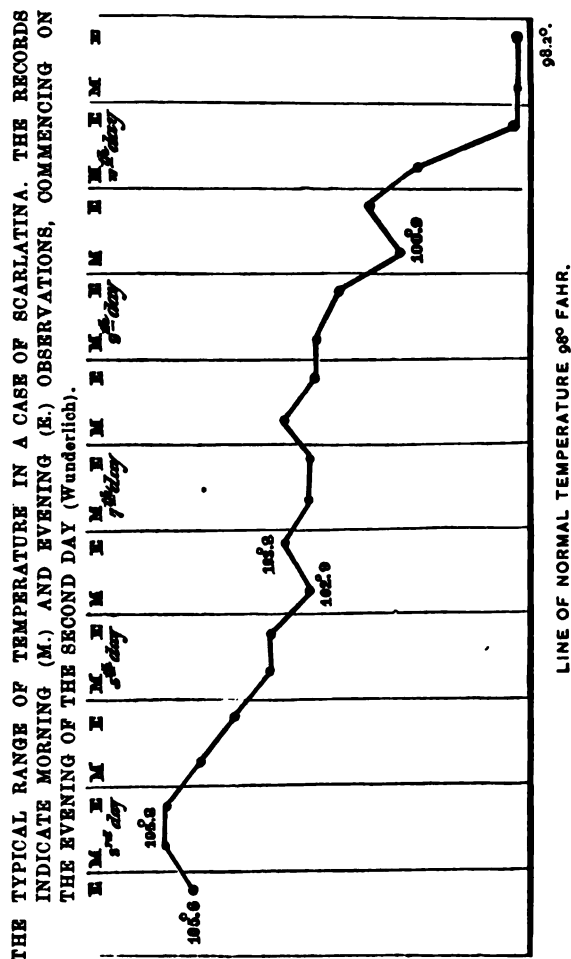
proaches yellow fever and cholera. Again, the rule that the great specific action of the poison is expended on the skin, causing the specific eruption, has very few exceptions. Of this eruption there are several forms, such as *smooth*, *papulose*, *phlyctenoid* or *vesicular*. These are all evanescent after death.

In the smooth eruption the surface of the inflamed skin presents no inequality either to the sight or touch. The *scarlatina papulosa* has an eruption in which the papillæ of the skin are enlarged, and the appearance is that of roughness, or "goose-skinned." The third form is when the eruption is accompanied by a number of vesicles filled with serum, which ultimately shrivel up and desquamate.

Whatever the ultimate form of the eruption may be, its first appearance is by innumerable small bright-red puncta, dots, or maculæ, separated by interstices of healthy skin. These puncta or maculæ are at first very minute points all over the affected parts of the skin, which are usually more or less rough to the touch; but they quickly become confluent, so that in a few hours the redness becomes general over the parts attacked. The color, in ordinary cases, is in the first instance a bright-red, like that of a boiled lobster, but on the decline of the disease it becomes deeper, and more resembles that of beet-root, while in severe cases it is of a pink blush, rather than a scarlet efflorescence, or it may be livid, and intermixed with petechiæ. But whatever tint the eruption may assume, it has this peculiarity, that it disappears on pressure, and again returns from the periphery to the centre on that pressure being removed. The color is also always brighter and more vivid in the flexure of the joints, and about the hips and loins, than over the rest of the body. A sign of scarlatina, in connection with the eruption, has been described by Bouchut, as pathognomonic. It consists in an enduring white stripe, produced by pressure with any hard substance on the skin occupied by the eruption. This phenomenon is ascribed to an increase of the contractile power of the capillaries, and which is proportionate to the intensity of the disease, the regularity of the eruption, and the amount of vital power (*Syden. Society Year-Book*, 1861, p. 130). The termination of this inflammation is generally by desquamation of the cuticle, and the desquamation begins with the decline of the eruption; and is usually completed by the end of the second week, unless it is longer delayed by successive crops of eruption, and consequent succession of exfoliations. There is no fever in which this phenomenon is more conspicuous. A few days after the commencement of the desquamation, albumen may be detected in the urine in small quantity, which continues to be given off for several days, along with a considerable amount of epithelium from the uriniferous tubules (DR. J. W. BEGBIE). There are also cases in which the albuminuria is associated with *anasarca*; and three stages have been recognized in which this complication occurs: (1.) A febrile stage, characterized by fever of an intermittent character, and by rapid serous extravasation and infiltration; (2.) A chronic stage, in which the

affection follows a slowly progressive course; (3.) A period of resolution (HAMBURGER). Such cases sometimes terminate by uræmic symptoms and convulsions. An unusual case of this nature has been recently recorded by Biermer. It happened with a boy five years and a half old, and ended fatally on the thirty-fifth day. No urine was passed for 108 hours between the twenty-first and the twenty-sixth days of the disease, and extremely little for five days more. Yet during these ten days there were no uræmic symptoms, nor any notable dropsy. The uræmic symptoms first set in after the urine began to be secreted freely, and it was but slightly albuminous (*Syden. Society Year-Book*, 1861, p. 218). Occasionally the *squamæ* of the cuticle are so large as to preserve entire the whole epidermis of the palms of the hands and of the soles of the feet. Frequently, however, the material of desquamation is furfuraceous or scaly. Frank has seen the cuticular *squamæ* come away with the hair, nails, and even with *verruæ* attached. In a few instances the termination is by ulceration and sloughing of large portions of the integument.

Whatever may be the color or description of the eruption, it does not attack all parts of the body simultaneously, but appears partially, or in a succession of crops, the order of which may be stated as follows: On the first day it spreads universally over the face, neck, and upper extremities; on the following day over the trunk, but is less general on the back than on the abdomen; and, lastly, on the third day, it has extended itself over the lower extremities. The duration of each crop is about three days, when it disappears, and in the order of attack, falling from the head and upper extremities on the fourth day; from the trunk on the fifth day; and from the lower extremities from the sixth to the eighth day. The order of attack, however, which has been mentioned is not constant, for in some few instances the eruption appears first on the trunk and lower extremities, and only on the second day very faintly on the face and upper extremities. The disease attains its height, and the fever maintains its course, usually from the fifth to the ninth day, when, in favorable cases, continuous defervescence sets in, and all the symptoms begin to decline. The fever does not subside on the appearance of the rash, as is the case with small-pox, but continues with various degrees of violence and ranges of temperature, throughout its progress. The pulse is often 120 to 130 in a minute, and sometimes beats with considerable force. The skin frequently indicates by the thermometer a temperature of 105°, 106°, or even 112° Fahr.; and it is dry, with a sensation of burning heat till about the third day, when the maximum of temperature is attained. From the third to the ninth day the range is maintained between 103.8° and 102.9°, and begins to subside about the tenth or twelfth day, after which the defervescence is continuous. The difference in these respects between scarlatina and measles may be appreciated at once by glance at the account and the diagram given of measles, and comparing it with the following, which shows



There is no remarkable increase of fever heat preceding complete defervescence; and after the *exanthema* has reached its maximum, the decrease of temperature proceeds by no means rapidly. The commencement of the decrease may be marked by a few decided and rapid falls; but its farther fall is decidedly lingering, and is even sometimes interrupted by small increases of temperature, so that the whole process of defervescence occupies, as a rule, from five to eight days. It is only in very mild or anomalous cases that the temperature rarely exceeds 101.8° Fahr., and these cases sometimes show a rapid defervescence, completed in a single night. It is essentially a short fever, the range of temperature, according to Dr. Sidney Ringer, forming cycles composed of a variable number of days, generally of five; a fall of temperature taking place on the fifth, tenth, or fifteenth day of the disease (*Med.-Chir. Society Trans.*, 28th January, 1862).

The poison of scarlatina as frequently falls on the mucous mem-

brane of the eyes and nasal fossæ, and excites a similar eruption over those parts, as on the skin, at first consisting of a distinct punctated or dotted appearance, which changes in a few hours to one of diffused redness. The inflammation of the ocular membrane, however, has this peculiarity, that it does not distress the sight, for the eye bears light without inconvenience, and in no case is it combined with coryza. Neither is sneezing a consequence of the affection of the nasal membrane; and only in a few severe cases is there any discharge from the nostril. As the eruption attacking these parts generally appears with the first crop of the *exanthema* of the skin, so does it generally die away with the cutaneous eruption. This inflammation usually terminates by resolution; but in a few instances the alæ of the nose ulcerate, and sometimes mortify.

The lingual and buccal mucous membranes are also often the seat of a similar exanthema, presenting nearly the same appearance as in other parts. The papillæ of the tongue, however, are singularly elongated and enlarged, and stand up salient and erect, and of a deep scarlet color, above the thick, white creamy mucous fur which coats the lingual membrane; and hence the term "strawberry tongue," from the supposed resemblance to the exterior of a strawberry. The tip of the tongue is of a vivid red, through development of the papillæ. By and by the fur falls off, and the whole dorsum of the tongue is then left clean, red, and raw-looking. This affection lasts longer than that of the eyes and nose, and usually terminates by resolution, although, in a few instances, the buccal membrane ulcerates and mortifies.

The sore throat, or inflammation of the faucial membrane, though not so constant an affection as that of the skin, yet when it does exist, it is often of much longer duration, and is a much more grave disease. It may either precede all the other symptoms, or it may occur at any period of the fever. This inflammation, at first punctated, then diffused, usually runs into ulceration, and the character of the ulcer is so completely in unison with the state of the constitution as to enable us, according as it is slight or severe, to divide scarlatina into two great varieties—namely, into "*scarlatina mitior*" and "*scarlatina gravior*." The first, or sthenic form, is marked by a greatly enlarged or swollen state of the tonsils, which are of a vivid or bright red-color; and, when ulceration takes place, the ulcers are seldom deep, or the sloughs slow to come away, but usually they separate about the fifth or sixth day; so that in mild cases the sore throat is healed about the eighth or tenth day, and in more severe ones about the fifteenth or twentieth. In malignant cases, or in *scarlatina gravior*, the tonsils are much less tumefied and enlarged, but much more loaded with blood, and of a deeper, and sometimes of a livid color. The ulcers, also, are deep and formidable, and the sloughs are thrown off later in the disease. They are likewise slow to heal, or not to the end of three weeks, and in severe cases not till four or even six weeks have elapsed, during which period the fever continues, and the patient remains in considerable danger.

The inflammation of the throat is not limited to the tonsils, but

extends over the fauces generally and uniformly, or symmetrically on either side, as is common with blood diseases. It may extend to all the neighboring parts, and an abscess may form in the pharynx, or pus may issue from the ears. The tympanum has been eroded, and in a few instances the inflammation has extended to the larynx, and the patient has died of croup. Besides these disorders, the glands of the neck often enlarge and occasionally suppurate, and, singular to say, sometimes not till after the sore throat has healed, and sometimes when there has been no previous affection of the throat, as if these parts were the seat of a specific action of the poison. Glandular swellings on both sides of the neck are not unfrequent, and have been described as "the scarlatinal bubo" (TROUSSEAU). They may be the best and most accurate index of danger in the later period of scarlatina, in so far as the danger depends on the sore throat and on the putrid infection of the blood (septicæmia) which accompanies it (W. T. Gairdner, *Clinical Medicine*, p. 193).

The inflammation of the cutis, as also of the buccal mucous membrane, is usually accompanied by some inflammation of the submucous or subcutaneous areolar tissue. This affection takes place as soon as the rash appears, and causes the hands to swell, so that the patient is unable to bend his fingers, and his face becomes tumefied and painful. In mild cases, however, the serum effused is absorbed, and the disease terminates without any unpleasant consequences; in severe cases it has a tendency to terminate in ulceration or in mortification. In children the toes of one foot have been known to slough off; and in some the integuments of the leg have mortified from the knee to the foot; while in others, mortification, commencing in the upper lip, has been known to spread till one-half the cheek was eaten away. Some have been known to die of mortification of the rectum, and others of a similar affection of the pudenda.

Such are the primary and secondary affections of scarlatina; but this poison has also some tertiary actions, giving rise to dropsy, as well as affecting the synovial membranes of the joints.

The dropsy which sometimes occurs after scarlet fever usually commences between the fifteenth and twenty-third days of the disease, and, almost uniformly, not till after all the other symptoms have subsided. The patient is liable to it during desquamation, as already mentioned, and for a considerable time afterwards. It begins with anasarca of the face, and afterwards of the hands and feet. In some instances the anasarca is universal, the whole areolar tissue of the body filling so rapidly as sometimes to destroy the patient in a few hours, the cavities of the chest and abdomen frequently filling at the same time. According to the observation of Dr. Wood and many others, it has occurred more frequently after mild than severe cases. Its forms are, therefore, *anasarca*, *ascites*, *hydrothorax*, *hydro-pericardium*, and even *hydrocephalus*; but, in whatever form, heaviness approaching to stupor is a common attendant. During the progress of the fluid effusion, œdema of the glottis must be watched for and relieved. The dropsy is generally accompanied with scanty and albuminous urine; and although the

presence of albumen, without diminished secretion, is almost a regular phenomenon in the course of the disease, independent of dropsy, as shown by Dr. James W. Begbie, yet if the urine becomes highly albuminous and diminished in quantity, the dropsical complications may be apprehended (*Edin. Med. Journal*, Jan., 1849, and Oct., 1852).

More or less congestion of the kidneys occurs in every case of scarlet fever, although, like the sore throat, it may often be so slight as not to give rise to any prominent symptom (BEGBIE, ANDERSON). The scarlatinal dropsy is very generally considered as most intimately connected with the kidney disease; and when the kidney disease is well marked, the characters of the urine exactly resemble those in acute Bright's disease (PARKES). On the other hand, there is also evidence decidedly in favor of the opinion that albuminuria may be wanting in scarlatinal dropsy (see Parkes *On the Urine*, p. 264).

The condition of the urine in scarlet fever ought to be ascertained daily in every case, especially during the period of convalescence. "It is of more importance," writes Dr. Andrew Anderson, "that you should examine the urine than that you should feel the pulse of a convalescent from scarlet fever." The urine has the ordinary febrile characters. During the first six days the amount is small; the urea and uric acid are increased in amount, and sediments of urates occur. The chlorine is sometimes greatly lessened, and augments during convalescence. On the sixth to the eighth day, if the case goes on well, the urine becomes abundant, pale, and the reaction neutral or feebly acid. There is bile-pigment present during the first six days; and in a large proportion of cases, though not in all, the urine becomes albuminous. Dr. Warburton Begbie believes it to be present at some period in almost every case. It is usually associated with a large amount of renal, pelvic, and bladder epithelium, but not with renal cylinders (BEGBIE), unless there be dropsy. The albuminuria occurring during desquamation is usually transient; but it may continue till an attack of dropsy occurs—disappearing and reappearing, when dropsy comes on a fortnight or three weeks later. In malignant scarlatina, as in malignant variola, there may be considerable hæmaturia or passage of dissolved hæmatin (Parkes *On the Urine*, p. 263).

Intercurrent inflammations of the synovial membranes have been described by Withering, Sennertus, Heberden, and others. This disease may attack the wrist, ankle, or knee-joints, and usually terminates by effusion of serum; and in some cases the cavities of the joints contain pus. This inflammation seldom occurs till after the eruption has subsided, and is generally a tertiary phenomenon in the course of the specific disease.

Such are the morbid phenomena which have been observed in the ordinary course of scarlatina, and with sufficient constancy to mark the disease as due to a specific poison; but these appearances are only to be found when the disease is of moderate intensity and the patient survives some days. In severe and rapid cases the patient may die, not from any organic lesion, but from the intensity of the shock, in the first instance, on the nervous system; for Bre-

tonneau, Tweedie, and Sims, all speak of having examined the bodies of persons who have died early in the disease, in which there was scarcely any appreciable lesion—coma, or other violent cerebral affection, carrying off the patient.

Although several varieties of scarlatina are described by authors, it is not to be supposed that they are equally distinctly defined in nature. Yet it not unfrequently happens that the characters of each variety are tolerably well marked. The following may be distinguished, namely: (1.) "*Simple Scarlet Fever*;" (2.) "*Anginose Scarlet Fever*;" (3.) "*Malignant Scarlet Fever*;" and (4.) "*Latent Scarlet Fever*."

Scarlet fever, of whatever description, essentially consists of fever, already described, and certain local inflammations; but among its more striking phenomena is the sudden and remarkable depression of the mental and physical powers of the body which the poison produces—a depression so great as sometimes to cause the death of the patient in a few hours, without any reaction, or any very sensible local lesion of the throat or other part being discoverable after death. On the contrary, there are a few instances in which the reaction is so great as to destroy the patient in an equally short time, and with a similar absence of all pathological phenomena; the affection of the skin being suppressed, and the sore throat wanting, the patient dies as if from the influence of an overwhelming poison.

The symptoms of scarlet fever under ordinary circumstances may be divided into three stages. The first stage occupies the period from the commencement of the disease till the appearance of the eruption, and is technically termed the "*primary fever*;" the second stage, that from the appearance of the eruption till its entire subsidence; while the third stage is reckoned from the disappearance of the eruption till the termination of the disease. The duration of the first stage is one, two, or three days; that of the second from six to eight days; while the third stage may either not exist, or vary from a few hours to two or three weeks, making the whole duration of the fever to vary from eight to thirty or more days. These stages are not, as in typhus, usually marked by changes of the tongue; for, except in the more severe forms of the disease, it continues coated with a white creamy mucus throughout the whole course of the disease. In "*scarlatina anginosa* or *maligna*," however, it becomes brown or black in the second, or at the commencement of the third stage.

The primary fever may be sudden in its attack, or the patient may complain for some days of slight indisposition. The early symptoms, whatever be the variety, are headache, pains in the back and loins, loss of appetite, sickness, and white tongue. The disease is, indeed, usually ushered in by vomiting—sometimes by very obstinate and troublesome vomiting. In slight cases it is sometimes the only noticeable symptom (ANDERSON). Still there are symptoms which distinguish it from other continued fevers; for the pulse, instead of being full and strong, is small and weak and rapid; the heat of the skin is more ardent; and, with such ranges of temperature as have been already noticed, these phenomena continue

through the whole course of the disease. The fever, however, varies greatly in intensity, as already indicated, from a mere febricula to the severest forms of a typhoid type in protracted cases.

1. *Simple Scarlet Fever.*

This form is known by the name of *S. mitis* and *S. sine angina*. It is the simplest form of scarlet fever, and is limited to cases with the fever and eruption, without any affection of the throat, or to cases with "a scarlet rash, with redness of the throat, but without ulceration."

The symptoms of this variety are extremely mild, so that the patient is frequently not confined to bed. The primary fever, except that the pulse is rapid, is little more than a mere febricula, and is not aggravated on the appearance of the eruption. The eruption appears at the end of twenty-four or forty-eight hours, and the crops follow each other according to the usual order of succession, appearing first on the face and neck and upper extremities; on the following day on the trunk; and on the third day on the lower extremities, when the disease has reached its acmé. On the fourth day the rash begins to decline, and fades from the face, neck, and upper extremities; on the fifth day it disappears from the trunk; and on the sixth or seventh day it is evanescent over the whole body. The color of the rash is always more florid during the night than in the day; and on its declining, desquamation takes place. With the disappearance of the rash the fever of this variety ceases, and the disease terminates; but it often leaves the patient in a state of considerable debility for several days, and may be followed by albuminuria.

2. *Anginose Scarlet Fever.*

In this form of the disease the specific action of the poison is mainly limited to one region—that of the throat—the eruption on the skin being altogether wanting, or appearing at a later period than usual, generally by one day; and, as a general rule, is less copious and less diffuse than in the other forms. It is "a more severe form of the disease, with redness and ulceration of the throat, and a tendency to the formation of abscesses in the neck."

There is seldom a season in which scarlatina has been in any degree epidemic, that cases have not occurred in which patients (not having previously had scarlet fever) are seized with severe fever and sore throat, unaccompanied by any eruption, and who, on subsequent exposure to the contagion of scarlatina, have been found insusceptible of the action of the poison. Hence it is inferred that the disease they have passed through must have been a variety of scarlet fever, or *scarlatina sine eruptione*, making itself manifest by a peculiar sore throat, associated with the febrile phenomena.

This disease, therefore, essentially consists in fever and sore throat. It has been stated that the state of the throat is constantly in unison with the state of the constitution, and consequently this form of disease, according to its severity, assumes all the symptoms which

accompany scarlatina simplex, or the more severe forms, with the exception of the absence of the eruption. It seems unnecessary, therefore, to give a separate detailed account of this variety.

In its milder form the essential character is, that the secondary or specific actions of this poison fall on two tissues—on the skin, and on the mucous membrane of the eyes, nose, mouth, and fauces. The fever which precedes the eruption in such cases lasts from twenty-four to seventy-two hours. The symptoms, however, are more violent than in the preceding species; for nausea or vomiting, great restlessness, headache, and some delirium frequently occur as early as the second day. The heat of the skin also is more considerable, and often raises the thermometer as high as 105° , while the pulse is quick, feeble, and fluttering, and shows the extreme debility the poison has occasioned. The primary fever having lasted its period, the specific actions of the poison are set up, and the eruption runs the course which has been described in scarlatina simplex, but its color is more intense, its duration more variable, and its attack more partial.

The *angina*, so marked a symptom in this affection, may precede the primary fever, may commence with the eruption, or may occur at some later day in the disease. It has many grades, and in this form of scarlatina they are all of the athenic or inflammatory type. Thus, in slight cases, the throat has merely the sensation of roughness, with some pain in deglutition; at a higher degree the tonsils are enlarged and ulcerated; while in cases of still greater severity they are swollen to a degree almost to occlude the fauces. In this latter case the act of deglutition is not merely painful, but in many instances impossible, and is impeded by a thick viscid mucus, which frequently requires the effort of vomiting to remove. The irritation of the fauces is sometimes propagated to the larynx, and the patient's voice is hoarse or inaudible, and perhaps he may ultimately die from this new affection. The parotid and submaxillary glands often enlarge, sometimes previously to the sore throat, more commonly about the fifth day, and again after the sore throat has healed.

The degree of fever is usually proportioned to the severity of the angina, and is accompanied by headache and sometimes by delirium. It does not abate on the appearance of the eruption, but continues till the throat is healed. If the sloughs come away early, or on the fourth or fifth day, the throat heals, and the fever perhaps subsides within a day or two after the eruption. It sometimes happens, however, that the sloughs do not separate till the fourteenth or fifteenth day; and in this case the fever runs on with equal violence after the disappearance of the eruption, and the whole disease is sometimes prolonged for three weeks or a month. In this case the tongue may become brown or dry, but it seldom continues so for more than a few hours. Observations as to temperature ought to be regularly and continuously made.

In the more severe forms of the *scarlatina anginosa* (and which have been described by some authors as the "*scarlatina gravior*"), the specific actions of the poison are the same as in *scarlatina mitior*, but the symptoms, both local and general, are more severe, and the

tertiary affections more frequent, and, consequently, the disease is more grave and the danger more formidable.

The more remarkable symptom which distinguishes this form of the disease is the state of the tonsils. In the milder form previously noticed, it has been stated that the tonsils are either slightly affected or greatly enlarged, of a bright red, and the ulcers comparatively superficial; but in this severer form the tonsil, though less swollen, is more gorged with blood, more livid in color, while the ulcers are foul, deep, and burrowing; the secretions of the mouth are more copious, and generally impregnated with the offensive sordes of the sloughs; while deglutition, if less difficult, is perhaps infinitely more painful, and the mouth often so tender that the slightest touch excoriates it. The ulcers likewise are slow to granulate, and only heal after a tedious treatment; and in the worst cases they spread in every direction, the parts tending to vesicate and even to mortify previous to the death of the patient.

The eruption offers some peculiarities, being often later by some hours in coming out, its color darker and more livid, its duration more uncertain, and its distribution more irregular and capricious than in the milder form. The primary fever, likewise, is usually longer, the delirium earlier, and the depression more complete than in the milder forms; and towards the close of the disease the tongue becomes brown, and the symptoms closely resemble those of the last stage of typhus fever.

Such are the more marked characters of the severer form of scarlatina; but it often happens that the progress of this disease (unless the range of temperature is regularly and continuously recorded) is silent, slow, insidious, scarcely marked by any prominent symptom till the degree in which the constitution is subdued by this formidable poison is shown by the inflamed nasal membrane discharging its fetid ichor, causing mortification of the alæ of the nose, or mortification of the lip or cheek; or it seizes on some remote part, as the toe, the leg, or the whole of a lower extremity, and which, for the most part, terminates the life of the patient. It may pass into the next form of the malady, namely,—

3. *Malignant Scarlet Fever.*

This form is that which is known as the “malignant sore throat,” or “putrid sore throat” of some authors; and is the name now generally applied to certain cases of extreme severity, into which some of the forms already described may pass, as if by insensible gradations. In this variety “the throat tends to slough; the scarlet rash is scarcely, if at all, visible, petechiæ are often seen on the surface, and the fever is of a low form.” In others, the violence of the attack is so sudden that the patient is at once struck down by the force or virulence of the poison, the type of the attack being at once septic, adynamic, typhoid, and malignant. The extreme severity of the constitutional symptoms is marked by the smallness, feebleness, and irregularity of the pulse; the oppressed, short, and quick respiration; the appearance of early raving, stupor, and some-

times coma, alternating with fretfulness and violence, dulness, and suffusion of the eyes, flushing of the cheeks, and dark-brown furred tongue. The rash appears late, and is of uncertain duration, and soon assumes a dark or livid color, or disappears in a few hours, re-appearing again after several days, if life is so far prolonged. Aphthous elevations in the throat, surrounded by a livid base, also become dark, and, bursting, they expose a surface of an excoriated, dark, gangrenous appearance. The passages of the fauces are always clogged up with much viscid mucus or phlegm, which produces a rattling noise in breathing, and increases the pain and difficulty of swallowing. The discharges, often sanious, are remarkably acrid, which issue from the nostrils and posterior nasal passages, causing soreness, excoriations, and even blisters on the surfaces and orifices over which, or through which, they flow. To this source the diarrhœa may be ascribed, which is sometimes severe at this period, and generally adds greatly to the sufferings of the patient.

The severity of the symptoms may produce death on the second, third, or fourth day of the disease, as from gangrene occurring in the course of the œsophagus or alimentary canal. In other instances in which the early symptoms were not remarkably severe, the aphthous state of the throat has all at once assumed a sloughing aspect, and has carried off the patient at the close of the first week. When the disease is continued beyond this time, death is foretold by the rapid, small, and weak pulse; by the rapid, languid, and oppressed respiration; frequent fluid acrid discharges issue from the bowels, and blood may be discharged from the nostrils, mouth, throat, bowels, or even from the kidneys; petechial or purpuric spots appear on the skin; and the patient is at last destroyed with local manifestations of the morbid state in several different parts and organs.

4. *Latent Scarlet Fever.*

The marked prevalence of anasarca in children has led to the discovery that such children have had previous attacks of scarlet fever, in such a mild form that it has escaped detection. In such cases the constitutional affection of scarlatina has been produced, but without any rash or sore throat being observed. On the kidneys alone the poison makes itself felt, and the dropsy which ensues is more severe, complicated, and fatal than that which follows the regular forms of the disease.

Sequelæ of Scarlatina.—Under this head it is proposed to notice what may be called the "tertiary actions of the poison." The effects produced in this way are often called by the people the "*dregs*" of the fever. The principal source of some of these sequelæ is found to be the primary obstruction to which the functions of the kidneys are so liable.

Amongst the most important of these sequelæ are the effects produced by an extension of the original affection of the throat towards the internal ear, by the Eustachian tube. When this takes place it not unfrequently happens that the small bones of the ear are

completely destroyed, the tympanic cavity becomes inflamed, ulceration of the membrane takes place, and perforation follows.

This morbid state is most difficult to remedy: a chronic discharge from the ear is established, which is of the most offensive kind, and which may continue till the whole of the internal ear is involved in the destructive and inflammatory processes; till the delicate and soft tissues in the cochlea and semicircular canals are destroyed, and the petrous portion of the bone itself dies; till the mastoid process, with its capacious osseous areolæ, becomes the seat of an obstinate carious process; or even till the brain itself, or the membranes, are involved in the unhealthy inflammatory process. Such a combination of effects occasions great and protracted sufferings, and sometimes in the end a fatal result (BRUCE, ANDERSON). A similar inflammation may destroy the tissues in the back part of the pharynx, extending towards the base of the cranium and upper cervical vertebræ.

A frequent form in which the tertiary actions of the poison of scarlatina are manifested consists in inflammation of the joints, and dropsy; and it is singular that these diseases are more often set up after mild than after the more severe forms of this fever. In such cases, about the time of the disappearance of the rash, the joints of the wrists or fingers, of the knees or other articulations, become swollen and inflamed, and present all the phenomena of an attack of acute rheumatism. This affection keeps up the fever, and prolongs the whole duration of the disease for many days beyond the usual period.

Again, in a given number of cases, not exceeding three per cent. in general, but in different seasons, or under different treatment, sometimes amounting to twenty per cent., the tertiary action of the poison produces dropsy. This affection usually occurs about the twenty-second or twenty-third day, or about the time when the patient is convalescent, and more often after a mild than after a severe disease. Dropsy more commonly begins with pallor of the countenance, and with œdema of the face; then the hands and feet swell, and, in a few cases, the areolar tissue of the trunk and lower extremities becomes enormously distended. When the areolar tissue is thus slightly or more generally distended with fluid, effusion may take place into the cavities of the head, chest, or abdomen. When the brain is threatened, the effusion is commonly preceded by the usual hydrocephalic headache, by convulsions, and sometimes by blindness. Effusion into the cavity of the chest or of the abdomen causes the usual symptoms of hydrothorax and of ascites, which have been described. In the former instance, however, the watery fluid is sometimes poured out so rapidly as to destroy the patient in a few minutes or in a few hours.

The first appearance of the œdema or effusion is usually preceded or accompanied by an accelerated pulse, by the urine being scanty, commonly turbid, and passed with pain: the quantity, however, is shortly increased; and if examined when passed copiously, it is found to be of low specific gravity, or from 1.011 to 1.017, and to contain albumen, sometimes blood, renal epithelium, and cylinders.

Diagnosis.—The only diseases with which scarlatina can be confounded are the acute forms of roseola and measles. Roseola, though usually accompanied by fever and sore throat, is distinguished from scarlatina by the eruption being confined generally to the chest. The diagnosis between measles and scarlatina will be better understood after the next disease we have to notice has been described—namely, the hybrid form sometimes assumed by a concurrence of the two diseases; and by a careful study and observation of the ranges of temperature in each.

Cause and Propagation of the Disease.—The earliest source of the poison is distinctly traceable to Arabia; and the disease has now spread over the whole world. It prevails at all seasons of the year, is always in existence somewhere, and often epidemic. Scarlet fever has been found to spread more extensively, and with greater fatality, among the poorer than among the wealthier classes of society. Both sexes are attacked in nearly equal proportions. All ages are probably liable to the disease; but it is most common to childhood—the feebleness of this early period of life facilitating, perhaps, the reception of the poison; and as children grow older, the less liable are they to be attacked.

In a clinical essay on the *History of Scarlet Fever*, most carefully worked out by Dr. B. W. Richardson, it is shown that scarlet fever attacks most frequently in the third and fourth years of life. The chances of attack decline rapidly after the fifth year. The seasons also seem to influence its prevalence and intensity. The months of October, November, and December, furnish in England the maximum amount of the disease—the months of April, May, and June, the minimum. This disease being established, the patient generates a poison which may be communicated directly, or which may contaminate the atmosphere. The disease is eminently communicable, so that no susceptible person can remain in the same room, and hardly in the same house, without contracting it. The *infecting distance* is consequently much greater than in typhus. Indeed, it is necessary to break up every academic establishment in which scarlatina prevails; for it is hardly possible to isolate children in the same house or school, however large, so as to prevent the disease from spreading. That scarlatina is capable of being directly communicated is shown by the fact, that children have been inoculated with the serum found in the vesicles which sometimes accompany the rash, and have taken the disease; but the inoculated disease not having proved milder than the natural, the practice has been properly abandoned. Another proof of the directly communicable nature of scarlatina is, that it has often been propagated by *fomites*, as by the clothes and boxes of boys returning from school. Susceptible persons also sleeping in a room lately occupied by patients laboring under scarlatina, and before the furniture has been washed and the bedding and walls well ventilated, have often taken the disease. The virus is destructible by heat at the boiling-point, or it may be disinfected artificially, as by the fumes of *nitrous acid*.

Dr. Willan says, that out of 2000 cases that he attended, he witnessed no instance of a *second attack*. Still, there are some excep-

tions to the statement that an attack of scarlatina gives an immunity from a second attack. Dr. Binns has seen instances of scarlet fever occurring twice in the same person, while Sir Gilbert Blane met with an instance of its occurring thrice in a young lady, without the least suspicion of ambiguity or possibility of mistake in diagnosis. Dr. B. W. Richardson shows that it may recur once or even twice in the same person. But these events are rare; and death from a second attack is unknown as a fact.

Scarlet fever has often coexisted with the vaccine disease, and with erysipelas, and this poison is consequently capable of coexisting in the system, not only with those that have been mentioned, but probably with all other morbid poisons.

The poison of scarlatina is absorbed by the mucous membranes; and absorption is also evident from the fact of inoculation having been effected through the skin. Children have been born laboring under this disease.

The period of latency varies from a few hours to ten days. In one case inoculated by Rostan the disease appeared on the seventh day; and the specific poison is probably capable of communication from the patient to others as soon as the primary fever has formed, and perhaps continues to be so till the sore throat has perfectly healed, supposing that affection to continue after the eruption has died away.

Prognosis.—The mortality from scarlet fever varies greatly according to the season, and also, perhaps, according to the fatality of the epidemic. In some years the proportion of deaths is not greater than three per cent.; but Sir Gilbert Blane says his practice gave one in four. He was consulted probably only in the worst cases, for in the same year it appears, from the reports of other practitioners, the deaths varied from one in six to about one in thirty.

There is perhaps no disease in which the progress is so capricious: for it is found to vary with the several forms, types, complications, epidemic constitution, and with the treatment in a most remarkable degree. The mortality is greatest in the period of infancy and childhood—from one to five years. In relation to mortality, it seems second in this country as to severity, typhus fever standing first (RICHARDSON). It is twice as fatal in towns as in the country. "There is one condition in which the disease is almost invariably fatal; that is the puerperal state. No precaution ought, then, to be neglected, no precaution ought to be thought excessive, which tends to prevent a woman from receiving the poison of scarlatina while pregnant or recently delivered" (DR. ANDREW ANDERSON). Fever during the pregnancy most certainly ends in abortion and death. If the woman be recently delivered, the disease will be of the most malignant type, and almost always fatal.

Treatment.—Scarlet fever being evidently accompanied by many highly inflammatory symptoms, the practice of bleeding was adopted on the first breaking out of the disease, in all countries, and, according to Willan, *with the most disastrous results*. This mode of treatment was adopted by Morton; and he speaks of witnessing 300 deaths from scarlatina in a week. It prevailed down to the time of Huxham, who abandoned it, and introduced a treatment by bark. In

this manner an entirely opposite system of treatment has been gradually introduced, and the records of medicine enable us to state the results of these opposite modes: Of cases treated at the Foundling Hospital by bleeding in 1786, and of cases treated at the London Fever Hospital in 1829, in the same manner, it seems proved that one in six died after bleeding, while only one in twenty-two died after a milder, if not a directly opposite, mode of treatment; and the conclusion which inevitably follows is, that the chances of recovery are diminished by the practice of bleeding, nearly in the ratio of four to one as compared with the chances of recovery supposing the patient not to have been bled.

It remains now to give some general directions for the treatment, and to point out the circumstances in which bleeding, purgatives, wine, and tonics may be most advantageously employed.

It should be laid down as a maxim, that in scarlatina, medical advice ought always to be had recourse to; for the worst cases we meet with (as those in which mortification of the nose, cheek, or limbs sometimes takes place) are those in which the disease has, from its apparently mild character, been left to itself.

In the mildest form of the disease it is sufficient to confine the patient to the house; to enjoin strictly a milk diet; to regulate the bowels; and, above all things, to avoid the *nimia diligentia medicorum*. If anything more be done, a small quantity of wine and water, proportioned to the age of the patient, may be given.

A gentle emetic at the outset is believed to have a happy effect in modifying the future course of the disease. Ipecacuanha, with or without tartar emetic, is the best form for administration; and half an ounce of castor oil ought to be given after the action of the emetic had ceased.

Looking to the morbid condition of the blood, and to the tendency which exists to the deposition of fibrine in the right cavities of the heart, small doses of *carbonate of ammonia* (three to seven grains) administered every hour, or every three hours, as soon as the symptoms are decided, have been recommended (PEART, WITT, RICHARDSON). Or, the *liquor ammoniæ acetatis* may be used with an excess of ammonia, to the amount of from three to five drops of *liquor ammoniæ* added to two fluid drachms of the former in a liberal quantity of distilled water (RICHARDSON). It is important to administer these medicines in small and frequently repeated doses; and, if possible, to let the remedy be taken as a drink.

The treatment of the milder forms of the fever, when the tonsils are considerably enlarged, is first to tranquillize the stomach and allay its perverted action when vomiting exists, either by small doses of the sulphate of magnesia, or by the effervescing draught, medicines which, according to the state of the bowels, may be given every four or every six hours. The gum-resin of *guaiacum* is of great service in subduing the *cynanche tonsillaris*, and may be prescribed in the following formula:

R. Magnes. Sulph., ʒvj; solve in Aqua, ʒviij; adde Pulv. Guaiaci, ʒiiss; Pulv. g. Tragacanth. co., ʒij; *misce bene*. One-sixth part of this mixture may be given every four hours, till the bowels are freely moved.

As soon as this object is effected, and it is ascertained that the tonsils are still greatly enlarged and swollen, the practice (supposing the patient to be *an adult*) is to relieve them by the application of six to twelve leeches to the throat; and the bleeding may be further encouraged by the application of a poultice. The trifling loss of blood thus sustained does not impair the general strength of the patient, if it is done sufficiently early, while it greatly reduces the swelling of the tonsils, and may prevent them becoming permanently enlarged. Another advantage is gained by the application of leeches to the throat—namely, that they relieve the affection of the head; for we constantly observe that, in diseases depending on morbid poisons, the head symptoms are relieved by relieving the part specifically acted upon.

The tonsils having been thus relieved, the fever ought to be permitted to run its course uninfluenced by medicine, the patient being only refreshed by the occasional administration of the saline draught, so grateful to his parched mouth and feverish state. If stimulation be adopted in these cases, we are apt to bring back the tumefaction of the tonsils; while, on the contrary, if we take more blood, we hazard producing the more serious accidents incident to scarlatina. The medicines, therefore, that have been mentioned should be persevered in till the disappearance of the eruption, and till the healthy granulations of the throat, and the decline of the fever, give certain evidence of a state of convalescence. At this point some mild tonic medicine is desirable, and prepares the patient once more for the fullest enjoyment of health. This is the most successful mode of treating cases of scarlatina in its milder forms. With children, however, it is better to trust to the soothing effects of warm poultices round the throat, than weaken the child by loss of blood.

The severe forms are characterized by the less swollen state of the tonsils, and by their being more livid and gorged with blood; by the ulcers being deeper and more spreading; and by the slough being fouler than in the milder varieties. As there is a greater tendency of parts to run into mortification, the necessity of adopting a more stimulating plan of treatment, and one more calculated to support the powers of the constitution, is manifest, and experience has shown this view of the case to be correct. The administration of wine, and of the "*extractum carnis Liebigii*," should therefore be the basis of the treatment of such cases. The quantity of wine for an adult may be from four to six ounces in twenty-four hours, and for a child about half that quantity. The wine may be either port or sherry, and should be drunk in small quantities, mixed with two-thirds water; or it may be given with sago, arrow-root, jellies, or other slops. The earlier the wine is given in the disease the better, and when delirium does or does not exist; regardless, also, as to whether the tongue is moist and white, or brown and dry; and it should be continued till the patient is decidedly convalescent. Liebig's extract of flesh should be given like beef-tea, as a drink. While pursuing this plan, it is necessary that the patient's bowels should be attended to. The treatment by wine is often ex-

tremely successful; and, as it is in general pleasant to the patient, whether a child or an adult, it is seldom refused. In cases more severe brandy may be required, or carbonate of ammonia in liquor cinchonæ, chlorinated soda, or creasote.

It may be proper, before adopting any special continuous mode of treatment, to follow the emetic first given by a dose of calomel, as a purgative, and this especially with children, to be followed in six or seven hours by castor oil or magnesia; and the bowels are ever afterwards to be kept open by remedies suited to the state of the patient and the nature of the disease. The following are the principal indications which must guide the treatment: If there is much excitement of the system, depleting cathartics are to be given: if nausea and vomiting prevail, a Seidlitz powder is of service. If the discharges from the rectum are acrid and acid, with acidity of the stomach, magnesia is preferable; if there is abdominal pain, castor oil with opium (Wood).

Bleeding is not successful in combating affections of the larynx; on the contrary, the most beneficial mode of treatment appears to be that of moderately supporting the powers of the patient by wine and mild tonics.

Again, when the synovial membranes inflame, and the joints become enlarged and swollen, all stimuli should be withdrawn, and a moderate action of the bowels should be kept up by means of the sulphate of magnesia, with camphor mixture, or carbonate of ammonia; and if pain be severe, some sedative should be added, as the tincture of hyoscyamus in a dose of fifteen minims.

The more formidable affection in scarlatina is dropsy; and from the great tendency to effusion into the head and chest, an active treatment is necessary. We should have imagined that in dropsy, a symptom in most cases of great debility, and following a disease whose characteristic is great depression, bleeding would have been dangerous and improper; but experience has shown that bleeding by leeches over the region of the kidneys is often of service; especially if œdema appears in the face, and is accompanied by headache, some blood should be taken—from two to four ounces in the child, and from four to eight ounces in the adult. The good results of cupping are also very remarkable; and even of continuously hot poultices over the lumbar regions, when it is not thought advisable to take blood. By these means renal congestion is relieved, and the urine becomes more copious and less albuminous.

Diaphoretic doses of antimony, and moderate but not severe purging, may be had recourse to. The compound powder of jalap, or the bitartrate of potash alone in drachm doses three times a day, are among the most useful; or it may be given as an electuary, in which the *cream of tartar* is mixed with nearly an equal quantity of *honey, treacle, or marmalade*, and flavored, if necessary, with a few drops of peppermint oil; digitalis also is much recommended, but it does not appear to possess any specific virtue. Dr. Andrew Anderson recommends the use of mercury in the form of blue pill, given twice or thrice daily, with squill and digitalis, till the urine resumes its natural appearance. The patient must at the same time be well

fed; and preparations of iron may be given with advantage. The *muriated tincture* seems to have the best reputation; and the *iodide of potassium* in small doses is also useful. With this latter remedy the *syrup of the iodide of iron* may be combined, if it is desirable to continue the *chalybeate*, or syrup of the phosphate of iron in drachm doses.

Blisters, as a means of relieving the throat, are unnecessary, and are better omitted.

Gargles are unnecessary for children, for they cannot gargle; but they are of the greatest service, especially the deodorizing gargles or washes, when the patient can be taught to use them. A weak solution of chloride of lime, or of chlorine water, or of Condry's fluid, or of the permanganate of potash, is well adapted to such a purpose. But the following is recommended as the most effectual gargle:

Solution of peroxide of hydrogen (containing ten volumes of oxygen), six ounces; tincture of myrrh, an ounce; rose water, five ounces (RICHARDSON).

This gargle may be used at pleasure: it is refreshing to the patient, and removes the offensive secretions readily. In the case of young children, who are unable to use a gargle, the throat may be washed out, by holding the little patient with the face downwards, and by pumping the solution over the surface of the fauces through a bit of gum catheter from a double-acting India rubber bag (Richardson, *Clinical Essays*, p. 110). As an invariable routine practice, Dr. W. T. Gairdner strongly recommends that "*the patient inhale the steam of hot water from the beginning to the end of the fever; as long, at least, as the throat is sore.*" In slight affections it is sufficient to employ infusion of linseed in water, acidulated with nitromuriatic acid, weak solutions of alum, nitre, or common salt. When membranous diphtheritic patches are observed on the fauces, and the color of the mucous membrane is of a dark red, capsicum infusion, or powdered red pepper, is an excellent application (Wood); and in children who cannot gargle, it may be applied with a hair pencil. Solution of zinc, and nitrate of silver, are also of service.

These details are given because the physician must decide, upon the merits of the individual case, the nature of the treatment he will adopt. But it must be remembered that cases of scarlet fever, if left to themselves, with rest and careful nursing, will generally get well. The mere intensity of the fever is no ground for active interference by way of treatment, if the pulse is full and of good strength. Much is to be trusted to the shortness of the fever, remembering that there is no disease in which the patient is more apt to be delirious, with less danger, than in scarlatina (W. T. Gairdner, *Clinical Medicine*, l. c.).

Dietetic and Preventive Treatment.—The diet of the patient should be slops, light nutritious broths, and jellies. Fumigation will not, it should be remembered, destroy the miasmata in the sick-room; and, consequently, the doctrines of cleanliness, of ventilation, and of separation, are as imperative in this disease as in small-pox. We

cannot disinfect the walls of the chamber, nor the clothes of the patient, except by washing them, or exposing them to a dry heat exceeding the boiling temperature. In general, then, the chamber where the sick patient has lain should be whitewashed and well scoured after the disease has subsided, before any person susceptible of the poison be allowed to sleep in it. It is important to guard against cold during convalescence. Children are not safe till the desquamation of the skin shall have been thoroughly accomplished—till all the old cuticle has been removed, and till the skin has resumed its natural softness. Therefore keep the patient in bed till convalescence is perfectly established; and subsequently insist on his being confined to his room, or in a room whose temperature is not below 60° Fahr., till desquamation is complete. If the whole surface of the body is well rubbed over once or twice a day with common olive oil, or a solution of glycerine in water (one part glycerine to three parts water), the irritation of the skin will be soothed, and the process of desquamation facilitated; and as soon as the child has strength to bear it, he ought to have a warm bath every second night, in which he may be well rubbed over with oatmeal and bran.

The ears of patients suffering from scarlatina ought to be carefully watched; and any complaint of pain ought at once to suggest an examination of the ears. A leech or two and warm poultices, followed by blisters, if necessary, may subdue the lesions of the ears which are apt to supervene. No stimulating applications should be used, beyond the frequent syringing with warm water.

Different prophylactic medicines have been recommended; amongst which belladonna has had the greatest number of advocates; but its value has diminished greatly, as the weight of testimony is against its possessing any prophylactic virtues (Wood). [Dr. N. L. North, of Brooklyn, New York, has proposed and used the hyposulphite of soda as a prophylactic, and with reason to believe, successfully.]

HYBRID OF MEASLES AND SCARLET FEVER.

LATIN, *Rubeola*; FRENCH, —; GERMAN, *Rötheln*—Syn., *Feüermasern*; Ritteln, *Falschen Masern*.

Definition.—A specific eruptive disease, preceded by, and accompanied with fever, watery discharges from the eyes and nose, sneezing, and sore throat. The eruption appears on the third or fourth day, and consists of crimson stigmata, rapidly running together into patches of an irregular shape, with obtuse angles, and of sizes varying from a threepenny to a crown piece, according to the severity of the case. The eruption continues from six to ten days, and terminates in desquamation by furfuraceous scales.

Pathology.—Those diseases now fully considered in the previous pages—namely, small-pox, measles, and scarlet fever—have been termed exanthematous diseases by some nosologists, in consequence of their principal phenomena being a very marked eruption.

The Arabians first described them, and considered them as varieties of one and the same disorder. Many essential differences, however, were soon observed to distinguish small-pox; but the points of resemblance between measles and scarlet fever were so many that it was not until fatal accidents had occurred, from great error in confounding them, that their differential characters were remarked, and their separate identity established. Now it is a generally received doctrine that measles and scarlatina, in their essence and in their symptoms, present two well-defined states of disease. This is, indeed, one of the most indisputable facts in Pathology. By Schönlein, measles has been classified as a peculiar exanthematic form of catarrh; and scarlatina is placed amongst the group of erysipelatous diseases; while, according to the experience of Dr. Küttner, of Dresden, there are "androgynous" cases calculated to embarrass the most experienced "diagnostiker." Measles and scarlet fever were especially confounded under the common name of *morbilli*; and even as late as the middle of the eighteenth century, writers of the highest repute supported the identity of measles and scarlet fever (the *morbilli confluentes* of Sir William Watson).

All authors before Sauvages (1768) had used the term *morbilli* (the term now in use) to designate measles; but he adopted a new name, and called measles by the designation of "rubeola"—an innovation which has caused much confusion, having been adopted by some (such as by Willan and Bateman) and rejected by others. Hildebrand, following the old nomenclature, calls measles *morbilli* and scarlet fever *scarlatina*; and terms the disease now about to be considered *rubeola*, as has been done by Dr. Copland. The German authors call it *rötheln*, and by this name it was first described by a most distinguished and learned Scotch physician, Dr. Robert Paterson, of Leith, in 1840. He is the only physician in this country who has given an original description of the disease in the English language, his description of the disease being drawn from many cases of it which occurred in his practice.

A difference of opinion prevailed amongst authors as to whether or not this disease is of a distinct and specific form. Those who have most recently described it (Hildebrand, Paterson, and Copland) consider it to be a disease possessing characters common to both measles and scarlet fever, as well as characters peculiarly its own. In truth, it seems to be a hybrid disease, developed from combined poisons of the two fevers, measles and scarlet fever. Dr. Küttner, of Dresden, states that he has seen occasionally in the same individual portions of the skin presenting the scarlatina eruption, while in other parts the eruption of measles was to be seen. He thus recognized not only examples of transition, but he recognized cases which may be termed hybridous (*Dublin Hosp. Gazette*, 15th Dec., 1858; and Ranking's *Abstract*, vol. xxix, p. 20).

Symptoms.—The febrile stage of the disease varies, like all the diseases already noticed, not only in the severity of the symptoms, but also in the length of the attack when compared with scarlet fever. It usually commences with rigors, not severe, but continuous. More or less cough soon makes its appearance—of the same clang-

ing nature which is observed in the febrile stage of true measles—and is very shortly accompanied with itchiness, redness, and weakness of the eyes, lachrymation, frequent sneezing, and watery discharge from the nose. In persons more advanced in life, severe frontal headache is complained of, together with rheumatic pains, more especially in the muscles of the back and chest, nausea, and sometimes vomiting, together with constant drowsiness. The skin is hot and dry, with the pulse above the natural standard.

A greater or less number of these symptoms is always noticed; but, in addition, sore throat is a most constant one. This, in some cases, is extremely slight, amounting only to a roughness of speech and trifling difficulty in swallowing; but in others it goes on to severe inflammation of the *tonsils*, *velum pendulum palati*, and surrounding parts. This last inflammatory affection is, however, more severe during the eruptive stage. The sore throat is one of the most characteristic features of the disease, occurring in the slightest and most gentle cases (ROBERT PATERSON).

The odor given forth by patients under this disease is described by Dr. Heim, of Berlin, as similar to, but stronger than that which scarlatina patients emit, and has been likened to the smell of a place where fish is kept—in short, fishy.

When the febrile state now described has continued for three or four days, the appearance of an eruption is sudden and general. It breaks out all at once over the whole body, and consists of bright and thickly set stigmata, which appear on the trunk, but are more sparingly dispersed over the face and extremities. It assumes different aspects and degrees of confluence, according to the severity of the case. Its first appearance resembles measles, but the stigmata rapidly run together, and soon assume an irregular shape, with obtuse blunt angles. These irregular patches are of an intense red color towards the centre, being gradually shaded off towards the margins, which approach in color that of the surrounding skin. The size of the patches in ordinary cases seldom exceeds a sixpenny piece; but in the severe forms of the disease they run still further together, and are to be seen of the size of a crown piece. In such cases, which are usually of a malignant nature, the whole body may be covered over with patches, varying from the size of a sixpenny piece to a crown piece, thickly set together, and of an intensely dark color towards their centres. The eruptive patches are felt to be distinctly elevated above the skin—some more than others, and always greatest in the centre of the patch.

During the continuance of the eruption, the general symptoms already described are usually aggravated, and not unfrequently new symptoms are superadded. The sore throat becomes much worse. The hoarseness becomes so great as frequently to cause entire loss of voice, and generally more or less external tumefaction of the throat takes place. In severe cases this is great, and is accompanied with much redness and swelling of the throat internally. There is a total inability to swallow even the slightest portion of fluid, which generally regurgitates by the nose. A large secretion of mucus of a vitiated nature takes place, the cough is constant, and is rendered

doubly severe by the state of the throat. The pulse is very frequent; the skin hot and dry; and there is great restlessness, expressed by children tossing the head frequently from side to side, accompanied with frequent starting; and they are sometimes seized with convulsions. It is in this stage, in the worst forms of the disease, that death generally occurs, and that by coma. It may, however, take place either by suffocation from the large quantity of vitiated mucus, or by convulsions and subsequent coma. Vomiting is an occasional symptom during this stage, and, like convulsions, is sometimes seen in mild cases of the disease in children.

The eruption in mild cases, in general, continues distinct for from four to five days, during which time the other symptoms are going on favorably, becoming gradually milder as the period of the decline of the eruption draws near. In severe cases, however, the rash keeps its bright color and distinct form for a much longer period—*e. g.*, six, eight, or ten days.

The termination of the eruptive stage is, in some instances, marked by what is termed a distinct crisis,—such as the occurrence of copious sweating, deposits from the urine, diarrhoea, and epistaxis. Most commonly, however, there is no such crisis, but the eruption gradually fades, and the disease subsides.

As this happens, the desquamation by furfuraceous scales gradually ensues. This event is indicated by the appearance of scales towards the centre of the patches of eruption, to the margins of which they gradually extend, and soon spread over the whole body. The scales are small, and not unlike those of measles. On the hands and feet the scales are larger, but never reach the size of those of scarlet fever (ROBERT PATERSON).

Lesions Seen in Fatal Cases.—The accounts of these are few in number. They vary according to the period of the disease at which death occurs. Death most frequently happens during the eruptive stage, from coma, or from the affection of the throat and lungs. No morbid appearances of a uniform nature can be observed connected with the mode of death by coma; but when death happens from pulmonary oppression, the lungs are found much congested, the mucous membrane of the bronchia injected, with a copious mucous secretion. The throat presents very similar appearances to those which are seen in scarlatina,—great tumefaction, and dark coloration of the membrane lining the throat, dark aphthous spots, and large quantities of vitiated viscid mucus.

Diagnosis.—The accompanying febrile symptoms at once distinguish the disease from roseola, as also do the peculiar characters of the eruption. The only other affections with which it may be confounded are measles and scarlet fever. The following table points out the diagnostic marks more clearly by contrast than can otherwise be done, and shows that rubeola, r  theln, or the mixed disease, has every right to be considered as a distinct affection:

TABLE SHOWING THE MOST PROMINENT DISTINGUISHING CHARACTERS OF SCARLET FEVER, RUBEOLA, AND MEASLES.
(Paterson.)

SCARLET FEVER.	RUBEOLA OR RÖTHELN.	MEASLES.
<i>Symptoms of First Stage, or Premonitory Fever.</i>		
Rigors; nausea; sometimes vomiting, thirst, and heat of skin; with sore throat, hoarseness, and delirium, in the anginous variety of scarlatina.	Shiverings; nausea; rarely vomiting; itching; redness and pain of the eyes, with increased flow of tears; sneezing, and watery discharge from nose; cough, sore throat, and hoarseness.	Rigors; nausea, and sometimes vomiting; frequent starting during sleep; itching; redness; pain of eyes; watery discharge from eyes and nose; sneezing; harsh cough.
<i>Duration of Premonitory Fever.</i>		
Premonitory fever is of short duration; the eruption most generally making its appearance on the second day.	The eruption generally breaks out on the third or fourth day, so that the premonitory fever is prolonged over that time.	Eruption makes its appearance towards the close of the third, or beginning of the fourth day.
<i>Appearance of Exanthematous Eruption.</i>		
It first appears in innumerable red dots or points, being at first of a pale-red color, soon acquiring a deeper tint, and at last giving the affected portion of skin a uniform red appearance.	The rash appears in minute dots, and rapidly assumes the appearance of irregular-shaped patches, with obtuse angles, varying in size from that of a three-penny to much larger than a crown piece. The red rash is gradually shaded off with the surrounding skin.	The rash appears in minute red points, like flea-bites; several of them soon coalesce, and form rounded masses, irregular-shaped crescents, or semicircular patches.
<i>Roughness or Elevation of the Affected Skin.</i>		
There is a perceptible roughness in the skin affected with scarlatina. It is in general most evident on the breast and extremities, and seems to consist of the enlarged papillæ of the skin.	In this disease, more especially in the severer forms of it, the patches of eruption are distinctly and considerably elevated, and more especially towards the centre of the patch.	The elevation of the patches of eruption in measles is slight; though in general distinct in the worst cases, they are certainly not at all elevated as a rule.
<i>Part of the Body First Affected.</i>		
The efflorescence is first perceptible on the face, neck, and chest, gradually passing downwards, and becoming diffused over the whole body.	The efflorescence first appears on the trunk of the body, the whole of which it at once occupies. It is always more sparingly seen on the extremities, but seems to break out there at the same time as it does on the trunk.	The efflorescence first appears on the forehead and among the roots of the hair, and spreads slowly and successively over the neck, chest, trunk, and extremities.
<i>Duration of the Eruption.</i>		
The eruption remains present three days; begins to disappear on the fourth day; and is almost entirely gone by the termination of the fifth day.	In the röteln the duration of the eruption seems to depend upon the severity of the disease; in mild cases remaining out four or five days, and in bad cases six or ten days.	In this disease it remains out three days.

Symptoms accompanying the Eruption.

The symptoms which accompany the eruption in each of the three diseases are quite the same as those of the premonitory fever. It is proper here, however, to remark, that it is only in the anginose and malignant varieties of scarlatina that we have sore throat, there being little or none in the simple scarlatina, while in the mildest kind of r  theln this is always a prominent and troublesome symptom.

SCARLET FEVER.

The cuticle in this disease is thrown off in patches of considerable size, the largest being from the hands and feet.

RUBEOLA OR R  THELN.

Desquamation.

The desquamation of r  theln consists of minute portions of cuticle, like scales of fine bran.

The desquamation always begins towards the centre of the eruptive patch, and gradually extends to the circumference.

MEASLES.

The desquamation of measles consists of minute portions of cuticle, like scales of fine bran.

Sequel  .

Anasarca is the most common sequela of scarlet fever. It is extremely common, and most frequently occurs after the mildest cases; swelling and suppuration of the cervical glands is also common.

"I have noticed one case of dropsy after a mild, though well-marked attack of this disease; swelling and suppuration of the cervical glands also frequently takes place." (DR. PATERSON).

Affections of the lungs and pleura; tedious distressing cough; chronic bronchitis; pneumonia tubercles; gangrenous inflammation of cheeks, gums, lips, genital organs, &c.; dropsy occasionally occurs, but very rarely; diarrh  a is very common after some epidemics.

Prognosis.—It requires to be as guarded as in scarlatina; for, like scarlatina, rubeola is often an extremely and rapidly fatal disorder. The greater or less acuteness of the premonitory fever generally affords us a means of judging as to the probable severity of the eruptive disease; and in general it is a mild disease. To have a copious secretion of mucus in the back of the throat is always a bad symptom, or regurgitation of fluids by the nose. The chest ought to be examined from day to day, as sudden inflammatory action is apt to be established, and often it rapidly proves fatal. The condition of the urine requires also to be daily investigated.

Treatment.—The treatment is similar to that of scarlet fever. The functions of the skin are if possible to be stimulated; and Dr. Paterson found that the *aqua acetatis ammoni  *, in the proportion of two ounces to half an ounce of antimonial wine and four ounces of water, made into a mixture, was the most useful agent. The use of colchicum was also had recourse to with decided benefit.

DENGUE.

LATIN, *Denguis*; FRENCH, *Dengue*.

Definition.—A peculiar febrile disease, commencing very suddenly, and conjoined with severe pains in the joints, which swell; succeeded by general heat of skin, intense pain in the head and eyeballs, and the

appearance of a cutaneous eruption or efflorescence on the third or fourth day, commencing on the palms of the hands and spreading rapidly over the whole body. It rarely continues visible beyond twenty-four hours. Remissions and relapses are numerous; and the disease may persist about two months, marked by great prostration and cachexia. The relapses are marked by rheumatic or neuralgic phenomena. It occurs in the West Indies and elsewhere.

Pathology.—This disease seems to combine an exanthematous eruption ushered in by fever, with a rheumatic or neuralgic state; and the course of the malady is so divided by intervals and remissions as to give one the idea that relapses are of frequent occurrence in its course. It has been chiefly prevalent in Rangoon, Calcutta, Berhampore, Patna, Benares, and Chunaighur, in the East Indies; the Island of St. Thomas in the West Indies; the Southern States of America; the ports on the Gulf of Mexico; the cities of New Orleans, Savannah, Charleston, Philadelphia, and New York. It was epidemic in America in 1824–28; and nothing appears to have been heard of it again till 1847 and 1850, when it again visited the Southern States. An epidemic of dengue has been recently described by Lemmon as having occurred in Virginia (*Amer. Med. Times*, Feb. 16, 1861). It is not known as an epidemic disease in Great Britain. It has been described by Nicholson, Mellis, Kennedy, Wood, Cavell, Twining, Mouat, and Goodeve. Dr. Richardson in his admirable *Clinical Essays*, notices the circumstance that we not unfrequently meet with “scarlet fever connected with acute rheumatic fever;” and he ascribes the first notice of this connection in this country to Dr. Golding Bird. Subsequently the circumstance was noticed by Dr. Kelso, of Lisburn, and by Dr. Ross. Dr. Andrew Anderson, in his *Lectures Introductory to the Study of Fever*, notices that rheumatic pains of the arms and legs, often very severe, connected, as he supposes, with the poisoned state of the blood, are not unfrequently met with. Lastly, Dr. Richardson himself records the case of four children, in which the rheumatic state combined with scarlatina was distinctly expressed (*Clinical Essays*, p. 85); and Dr. Wilkes has noticed similar cases.

Symptoms.—The invasion is very sudden, and the development rapid. In the greater number of cases the first symptoms have been headache, with intolerance of light, restlessness, and more or less chilliness, debility, pains in the back, the limbs, and joints. The small joints swell, and there is soreness, with stiffness of the muscles. The skin soon becomes hot and dry, the pulse frequent, the face flushed, and the eyes red and watery. The tongue, though red, is usually clean. A rash or papular eruption sometimes appears, though not generally at this stage. Painful swellings in the lymphatic glands of the neck, axilla, and groins are common. The testicles also swell, and continue so till the subsidence of the other symptoms. The febrile state lasts from twelve hours to three or four days, after which it subsides, leaving the patient very feeble. This remission lasts for two, three, or four days, when a return of the fever and pains, with a thickly coated tongue, nausea, and epigastric tenderness, mark another phase of the disease. On the fifth, sixth,

or seventh day the eruption appears in the form of a scarlet efflorescence on the palms of the hands, which spreads rapidly over the body, and gives relief to the symptoms of febrile irritation. The eruption is extremely variable in character, being sometimes smooth, red, and continuous, as in scarlet fever; sometimes in patches, rough, and of a dark hue, as in measles; and occasionally either papular, vesicular, pustular, or furunculous; often with a mixture of two or more of these forms. The complaint gradually subsides, and leaves the patient with some rheumatic stiffness or soreness for a longer or shorter period, with feelings of weakness and mental depression. The duration of the affection varies with the length of the remission; but on the average is about eight days. Decided implication of the mucous membrane of the mouth and throat prevailed in the last epidemic in Calcutta, with an almost entire absence of the articular pains.

Treatment.—Emetics and purgation subdue the fever; but as the disease runs a specific course, time is an essential element in the treatment. Calumba, rhubarb, and soda form a useful alterative medicine. Ophthalmia is sometimes consequent on this disease, and is to be subdued by leeches to the inner membrane of the eyelids. Palliation and alleviation of symptoms, as they arise, chiefly by opium and alkaline remedies; following the indications given under “Scarlatina” and “Rheumatism.”

ERYSIPELAS.

LATIN, *Erysipelas*; FRENCH, *Erysipèle*; GERMAN, *Erysipelas*—Syn., *Rothlauf*; ITALIAN, *Risipola*.

Definition.—A febrile disease, associated with a peculiar eruption of the skin. The inflammation which attends this eruption is apt to spread indefinitely, and may involve the areolar tissue beneath the skin.

Pathology.—As in other diseases of the miasmatic order, it is believed that in erysipelas a specific poison is absorbed and infects the blood, and that after a given period of latency it produces fever. The specific action of the poison, however, is mainly made manifest by inflammation of the skin and subcutaneous areolar tissue, which runs a definite course, and sometimes terminates in inflammation of the membranes of the brain. The inflammation and the fever are of a peculiar nature, not yet clearly understood. In Scotland the disease is known by the name of the *Rose*; in England it is sometimes called *St. Anthony's fire*.

Idiopathic erysipelas is very constantly preceded by fever—eighteen times out of twenty—and although it may be supposed that the fever is consecutive to the inflammation of the skin, yet before the redness of the skin is seen, the temperature, if measured by a thermometer, will be found above 98.6° or 99° Fahr., and attended with general malaise. The affection of the areolar tissue may be trifling, but it is seldom altogether wanting.

The pathological phenomena which result from the action of the

poison on the skin are, first, that the cutis is diffusely inflamed, the affected part being either of a bright scarlet or a rose-colored tint, evanescent on pressure, but returning on that pressure being removed. This inflammation is usually of great extent, occupying very commonly the whole face, head, and neck, or a considerable portion of the trunk, or one or both lower or upper extremities. It runs a course which may be characterized as "tolerably regular and definite."

It may terminate by resolution, by vesication, or by gangrene. When it terminates by resolution, the rose tint gradually changes to a deeper and more venous hue, and at length fades away, leaving the skin of its natural color, but with the texture so impaired that desquamation follows. If the inflammation terminates in vesication, the cuticle is raised into a number of vesicles of greater or less size, and sometimes into large bullæ or bladders containing a yellowish transparent serum. The cuticle at length ruptures, the fluid is discharged, and a crust sometimes forms, which, on falling off, leaves the skin underneath either sound or superficially ulcerated. Should the termination be by gangrene, the skin becomes livid or black, its whole texture more or less disorganized, while the bullæ or phlyctenæ which often form in these cases are filled with a bloody serum. The cutis, when examined after death, whatever may have been the form of the disease, is always found greatly thickened and infiltrated, but the redness, except in cases of gangrene, has entirely disappeared.

It is seldom that erysipelas is limited to a simple affection of the skin. More commonly, at some period of the disease, the areolar tissue beneath the affected skin becomes the seat of a serous exudation; and it may suppurate, or proceed to gangrene. When the termination is by effusion of serum, the quantity of fluid effused is generally so considerable that the head, face, or limb, is greatly, and sometimes even hideously, swollen; and if the part be now incised, the vessels are seen enlarged and numerous, and the cellular tissue loaded with serum, sometimes turbid and flaky. The tissue is more easily torn than usual. This inflammation may terminate by absorption of the serum; but in a few cases ulceration follows, and in some gangrene. Adhesive inflammation seldom takes place in erysipelas without its being accompanied by a serous effusion and the occurrence of suppuration. Suppurative inflammation is, indeed, uniformly preceded by serous effusion, and the result may be the formation of an abscess; or, what is much more common, pus may be *infiltrated* through the areolar tissue, uncircumscribed by an adhesive inflammation—a circumstance improperly considered by some as pathognomonic of erysipelas. The parts more usually the seat of phlegmonous circumscribed abscess are the eyelids and the integuments covering the cheek-bones, and the pus in these cases is usually of a laudable and healthy character. In all other parts of the body the abscess is generally diffuse; and, the inflammation being of a low type, the pus is poor, and often little more than a fetid sanies. Should the parts slough, the purulent fluid becomes loaded with a dirty broken-down areolar tissue, generally mixed with some loose lymph. In some instances the

suppurative process extends between the muscles, causing extensive and often irreparable mischief. In the event of this inflammation terminating by gangrene, the integuments of an entire limb are sometimes detached, laying bare the muscles, a large artery, or a bone, involving the aponeuroses and tendons, and sometimes destroying the interior of a joint. Gangrene, however, does not equally take place in all parts, for it is seldom seen on the scalp, the face, or the trunk. It is the extremities, then, and more especially the leg and thigh, and also the labia and scrotum, that are apt to suffer from this affection.

The appearances found after death from erysipelas are similar in many respects to those found in cases of typhus fever.

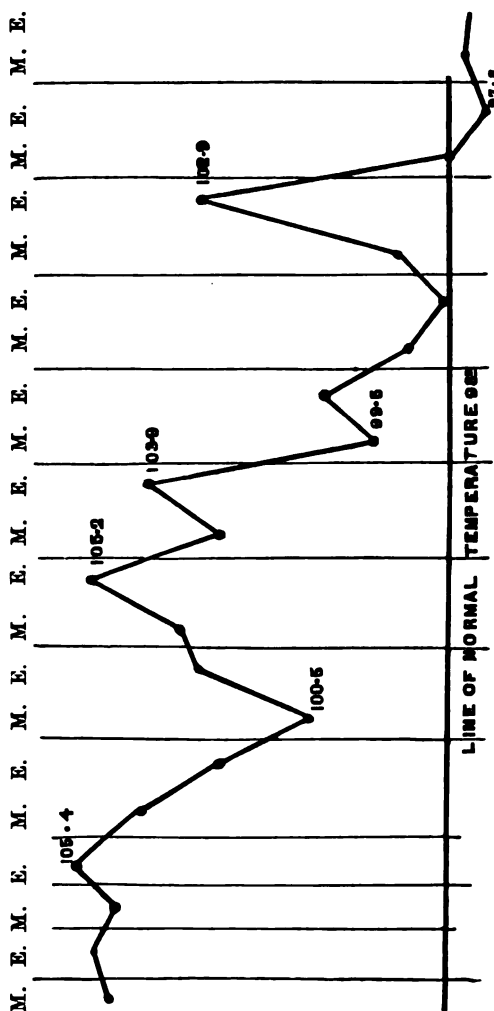
Symptoms.—The symptoms of erysipelas arise out of the fever and local affection, and appear of various degrees of intensity.

In acute sthenic cases the erysipelatous inflammation is preceded and accompanied by fever; and the attack may be sudden, or ushered in by rigors, irregular flushings, muscular pains, accelerated pulse, white tongue, nausea, vomiting, and deranged bowels. Sore throat is an early and constant accompaniment. These symptoms, when they do exist, last for some hours—perhaps till the end of the second night or beginning of the third day—when the fever becomes continued, the tongue brown and dry, and shortly afterwards the cutaneous inflammation appears, but without any remission of the fever. The inflammation generally appears at the seat of any injury to the skin, such as a wound, and is most intense there. By some, indeed (Trousseau, for example), it is held that erysipelas always originates from some external injury or irritation, which may be very slight. But this character erysipelas has only in common with other eruptive diseases, as Mr. Paget has described in his admirable Address on Surgery, delivered to the British Medical Association at their meeting in London, in August, 1862. He noticed that, “having cut a boy for stone, the boy became very ill three days afterwards, and seemed in danger of his life; but soon a vivid red eruption appeared at and about the wound. This was measles, earliest and most intense at the seat of injury, just as erysipelas might have been. Thence it extended, and ran its ordinary course, and did no harm.” Mr. Paget states that he has seen similar events with scarlet fever, the eruption commencing in an injured and inflamed knee. Dr. William Budd records similar events in a case of small-pox, in which the eruption first appeared and was most intense over a bruise on the nates. The argument from such facts is, “that the local determination of erysipelas, and of all other allied diseases, after operations, is no proof of their local origin or local nature.”

Diagnosis.—The diagnosis of erysipelas is, in general, easy. For a few hours, perhaps, if a joint be attacked, it may be mistaken for acute rheumatism; or if a surface be attacked, it may be confounded for a short time with erythema, but the intumescence and spread of the disease quickly enable us to rectify the error.

Frank has pointed out a symptom which he considers diagnostic—namely, that whenever a patient has exhibited, for twenty-four

THE FOLLOWING DIAGRAM REPRESENTS THE TYPICAL RANGES OF TEMPERATURE IN A CASE OF ERYSIPELAS WITH RELAPSES AFFECTING THE FACE. THE RECORDS INDICATE MORNING (M.) AND EVENING (E.) OBSERVATIONS.



The course of the fever in erysipelas is very similar to that of measles; but the advance of the fever to its height continues longer, and the epoch for the commencement of the defervescence vacillates between the *fourth* and the *eighth* days. The defervescence, as a rule, is rapid, the normal heat being attained, or nearly so, in from twelve to thirty-six hours. Frequently, however, the case is not terminated therewith. New relapses may take place, and the course

of the disease may be prolonged through two or even three weeks. These relapses are severally of short duration; but they come on again and again, and are ushered in by a smaller or larger increase of heat, and they are connected with a renewed spread of the cutaneous affection; and it is only after the eruption has ceased that complete and definite defervescence ensues. Very sudden changes of temperature are characteristic— 4° or 5° Fahr. in twenty-four hours, or a fall of 7° or 8° , commencing immediately on the appearance of the characteristic redness (COMPTON). This erratic and protracted form of erysipelas is most frequently met with in the aged, associated with gouty or rheumatic states of the system, as well as with albuminuria or renal disease. The *local* symptoms vary according to the part affected, the mode of termination of the inflammation, and also according to the character and duration of the fever.

When erysipelatous inflammation affects the face, it may begin either in the skin or in the subjacent areolar tissue. If the areolar tissue be primarily affected, the face at the inflamed part becomes swollen, but the skin suffers no discoloration for some hours, so that it is impossible to distinguish it from an ordinary attack of swelled face. At length, however, the skin inflames, and the part is now red, hot, and painful, as well as swollen, and the disease is fully formed. At the commencement of erysipelas of the face the attack is usually partial, and perhaps limited to the bridge of the nose, to one ear, to the lower eyelids, or to one cheek; but in severe cases it gradually extends, often involving the whole of the integuments of the face, head, and neck; so that at the end of three or four days those parts present a strangely swollen, disfigured, and even in some instances, hideous appearance, scarcely a feature being discernible. The nostril, moreover, is imperforate from internal swelling, so that the patient is obliged to breathe with his mouth open, while the inflammation may extend to the auditory passages, and render him completely deaf. Extension of the inflammation to the membranes of the brain sometimes takes place, while the external inflammation continues. This untoward event is followed by delirium and coma. But delirium frequently supervenes in the course of erysipelatous attacks, independently of any metastasis or extension of the disease to the membranes of the brain. It commences with wandering of the mind at night, similar to that which is observed in fever. Utterance is given for the most part to low, muttering, and rambling expressions, which rarely assume a noisy character, but which in fatal cases terminate by coma. When the patient has been of dissipated habits, or is otherwise of a dilapidated constitution, then the delirium resembles that of *delirium tremens*, not due to inflammation of the brain, but in consequence of an altered condition of the blood and of the nervous system (BARCLAY).

On the fourth, sixth, eighth, or some later day, the bright red color of the skin changes to a deeper hue; the serum effused is absorbed, desquamation takes place, and the skin gradually returns to its natural color. It is not unusual, however, for abscesses to form, particularly on the eyelids or cheeks, and which, being opened quickly, heal, and hardly retard the convalescence of the patient.

In some cases the disease becomes *erratic*, and extends over the chest or down the back, and desquamation is seen going on in one part while the erysipelas is spreading in another. This is characteristic of cases associated with gouty or rheumatic constitutions.

The extremities are more commonly the seat of erysipelatous inflammation than the trunk, and the lower extremities are more frequently affected than the upper. When these parts are affected, the fever is less severe than in erysipelas of the head; but the local symptoms are generally more formidable, for the degree of heat is greater, and the pain so severe that the weight of a sheet can hardly be borne. The inflammation likewise often involves the lymphatic vessels, when they can be traced by white or red lines for many inches, as from the knee or elbow to the inguinal or axillary glands; and these sometimes enlarge and suppurate. If the erysipelatous inflammation ends in suppuration, the abscess is always diffuse, and the swollen limb gives a peculiar sensation to the hand; and which has been compared to what a person feels with his feet on passing over a quagmire. The dark, black, discolored appearances of gangrene are too obvious to render any description of the parts so affected necessary. Numerous varieties of erysipelas are referred to in practical works, especially surgical, most of which are modifications of the disease as above described.

Besides the *erratic* form just noticed, there is the—

Phlegmonous Erysipelas (*Erysipelas Phlegmonodes*) and *diffuse inflammation of the cellular tissue*, in which the inflammation extends deeply into the subcutaneous tissues, and tends to spread indefinitely. It is attended with greater pain and swelling than the more superficial variety, and usually the general symptoms are more severe. Suppuration and gangrene of the areolar tissue are not uncommon; and if the disease penetrates beneath the fascia, the sufferings of the patient are greatly aggravated by the compression of the inflamed parts, and much organic mischief may result from the confinement of pus and the various products of the gangrenous state.

Gangrenous Erysipelas (*Erysipelas Gangrenosum*).—As the name implies, this form is accompanied with death of parts, and the tendency to death of tissue may be due either to the inherent depressing nature of the disease, or the depraved state of the system, as of the blood, the co-operating influences of an epidemic constitution, debility, confined and impure air, as in crowded hospitals, unwholesome or scanty food, or simply the excessive violence of the inflammations. The peculiar hot and burning pain, with the purple or livid hue of the redness, indicate the tendency to gangrene; and its near approach is shown by the slowness with which the blood returns after removal by pressure, and by the formation of vesicles (phlyctenæ) filled with turbid reddish serum. These vesicles are to be distinguished from those which are to be seen on the skin in severe contusions on fractured limbs. The fluid in the vesicles of gangrene can be pressed from under one part of the cuticle to another, which is not the case in the vesicles on a fractured limb or a severe bruise. Patients with typhoid fever, infants soon after birth,

and young children, are most frequently the subjects of gangrenous erysipelas; and it is not uncommon in hospitals, during the prevalence especially of malignant epidemics of erysipelas.

Cause.—The mystery which hangs over the origin of disease poisons does so, in a remarkable degree, over erysipelas; for this disease is often epidemic, and appears to be very constantly present in communities, and especially in large towns.

The predisposing conditions are age, mechanical or chemical injuries, as blows, punctured wounds, and incised wounds generally, bites of insects, or burns; also certain articles of diet, as mussels or periwinkles; and many diseases likewise, as dropsy with renal disease, typhus fever, and others of a debilitating kind. The effects of age in predisposing to this disease are considerable. New-born children, for instance, are occasionally subject to it, but from that period to adult age it is seldom witnessed. The period of life most subject to acute attacks is from twenty to forty; and to frequent asthenic attacks from forty to old age. Both sexes suffer in nearly equal proportions.

Propagation of the Disease.—The spread of erysipelas has been so frequently observed, both in the sick-room and in the wards of hospitals, that no doubt can exist of this disease being communicable by impalpable emanations. In the year 1760 erysipelas spread so extensively through the wards of St. Thomas's Hospital, in London, that it was believed the plague was in the hospital. Dr. Baillie described it as spreading also in St. George's Hospital, London; and Dr. Cullen, in the Hospital at Edinburgh. It has been found to spread extensively on board ship; and Drs. Wells, Watson, and others, have given several remarkable instances of its spreading in families. Dr. Steele writes, in his excellent *Annual Report on Guy's Hospital* for 1863, that "for some years past it has been customary to place patients suffering from erysipelatous wounds in these (the medical) wards, in order to diminish as much as possible the risk of extending infection in surgical wards, as well as to promote recovery in the patients themselves. It happened, however, that in one of the wards of the new hospital into which a patient suffering from erysipelas was placed in the course of the past year, *five* persons suffering from other complaints were attacked with the disease; and although none of the cases were attended with fatal consequences, the occurrence is sufficient to point out the danger which must be occasionally apprehended." Dr. Maclachlan, on the other hand, has never seen the disease propagated by contagion or infection in the infirmary of Chelsea Hospital; and he is disposed to think that the disease is less contagious or infectious when occurring in persons of advanced life than at other periods.

That it is communicable by some palpable virus, was shown by Dr. Willan, who says, that if a person be inoculated with the fluid contained in the phlyctenæ or vesicles of a genuine erysipelas, a red, painful, diffused swelling and inflammation analogous to erysipelas is produced. The danger, however, attending this experiment has not allowed it to be repeated.

Erysipelas also spreads by *fomites*. In hospitals, wards are occa-

sionally obliged to be cleared out, to stop the continued spread of this disease. In the navy the spread by *fomites* is so well understood that it is debated whether swabbing the decks or dry rubbing them is the best mode of disinfecting a ship, and preventing the spread of the disease. It has spread extensively, and for long periods, in the Birmingham, Edinburgh, Glasgow, and London hospitals, and is only got rid of by emptying and whitewashing the wards. It is said, however, that dry rubbing is preferable to washing, moisture appearing to promote the extension of the disease. The old "Dreadnought" hospital ship in the Thames was so impregnated with the *fomites* of erysipelas that she had ultimately to be broken up, and a new vessel substituted.

A patient having passed through an attack of erysipelas has no security against future attacks of the disease; and many persons suffer repeatedly from erysipelas; some periodically. There appears to be a constitutional predisposition to the disease in some people, and especially in those who have periodic attacks. Some women have attacks every month. Intemperance, and all influences which tend to depress the system, predispose to the disease, and hence partly the prevalence of the disease in hospitals. But there are unquestionably some unknown conditions of the atmosphere which seem to favor the dissemination of the disease. It has been observed that this predisposition to erysipelas exists in the ordinary wards of hospitals at the same time that puerperal fever prevails; and it was formerly not an unfrequent accompaniment of small-pox.

Period of Latency.—Erysipelas has occasionally followed a few hours after exposure to the infection. Dr. Elliotson thinks five days elapsed in his own case, and Dr. Watson has given three cases in which the interval was a week. It has been observed in hospitals that a fortnight has elapsed after its subsiding in one case and appearing in another in the same ward. It is probable, therefore, that the period varies from two to fourteen days.

Erysipelas and puerperal fever are interchangeable diseases, the one being able to induce the other by personal contact. Destructive epidemics of erysipelas have now and then occurred in Europe, and several parts of America have of late years been the scene of similar ravages, especially in the New England States, the South-western States, and the interior of Pennsylvania (Dr. Wood).

Prognosis.—The most experienced physicians consider erysipelas, at all periods of life, "a dangerous and deceitful disease;" and when it fixes on the face or scalp it is one of the most serious diseases to which an aged person especially is liable; for when all appears to be going on well, the membranes of the brain may become involved, or the powers of life may give way, the patient sinking suddenly under the depressing influence of the poison. The disease may extend to the fauces or the glottis, and the patient then dies suddenly from the œdema of the glottis which supervenes. The disease is peculiarly fatal to drunkards and to patients of broken-down habits; and frequently recurring attacks show such a bad state of health as indicates a speedy break-up of the constitution (MACLACHLAN). It is five times more fatal to people above

sixty than to people between fifteen and sixty years of age (*Registrar-General's Fifth Report*, p. 456).

Treatment.—Broussais states that when he served with the French armies in Italy, he has seen erysipelas allowed to run its natural course, and the result was, that it made immensely rapid progress, and ended either in suppuration, in gangrene, or in fatal visceral inflammation.

Erysipelas, in the opinion of some, is a disease of simple inflammation, and consequently ought to be treated by general and local bleeding; while, on the contrary, others contend that it is a specific inflammation; and long experience has shown that bleeding is often injurious, while a tonic mode of treatment is much more uniformly successful.

There are very few physicians, from the days of Hippocrates to the present time, who have not bled patients in erysipelas, and consequently this experiment has been made on a large scale; still, many of the warmest advocates of bleeding admit that the operation is occasionally followed by unpleasant consequences. Indeed, the treatment by bleeding has been often followed by so many unfavorable results, that many physicians, the most intelligent of the profession, affirm that, according to their experience, the practice is not only unfavorable but highly injurious. Andral is reported to have said, "In erysipelas with delirium, bleeding pales the skin, but the disease continues; the cellular tissue remains gorged, and death follows. We open the body, but find nothing." Cruveilhier says, "*des erysipèles rentrés*" is a consequence of unusual or too abundant bleeding, and he considers the question of bleeding in this disease to have been "*depuis longtemps jugée*." Blache and Chomel likewise say that "Experience has proved that general bleeding has no other effect than to blanch the eruption, without notably abridging its duration." In this country, Drs. Fordyce, Wells, Pearson, Heberden, and Willan, all give their testimony to the frequent ill effects of bleeding in this disease; and, in consequence, they, for the most part, recommend a tonic treatment.

It is therefore to be recollected that bleeding will not cure the *erysipelatous* inflammation, in the way that it produces a salutary effect on an idiopathic inflammation of the lungs, occurring in an otherwise healthy person. It is also to be borne in mind that as a rule, bleeding is not borne well by persons suffering from erysipelas; and it is necessary to be ever mindful of the fact that people of a certain class in populous towns cannot bear bleeding so well as those who pass their life in the country. For instance, a brewer's drayman in London, accustomed to rejoice in the beverage which he delivers to his customers, would sink suddenly under the influence of a bleeding: when if double or even treble the amount of blood were abstracted from a countryman suffering from erysipelas of a sthenic form, but heretofore in good health, it would produce but little effect, and that probably for good. Bleeding, as a rule, is only indicated in the young, the healthy, and the vigorous; and it must equally be avoided entirely in the aged and in broken-down cachectic patients.

Rest, saline laxatives, cooling drinks, and low diet, are the elements of treatment in mild and simple cases. An emetic is useful at the commencement; and I have seen, in the practice of an eminent surgeon, that frequent resolution of an erysipelatous attack has followed an antacid laxative, such as of rhubarb powders and carbonate of soda, together with the counter irritation of a mustard poultice over the stomach. Laxative and cathartic remedies are to be selected and apportioned according to the violence of the attack and its nature, as tending to the unfavorable results of the specific inflammation already noticed. Calomel is a most valuable purgative, as a sedative in febrile disturbance, especially when followed by castor oil, or the common black draught. The indications to the use of certain remedies, as given in the treatment of scarlet fever, are equally applicable here.

If the febrile state is not subdued, antimonials are of great service, for so far as they are diaphoretic in their action they tend to subdue the vascular excitement. If symptoms of nervous depression ensue, opium, or opium and ipecacuanha are indicated, also wine and quinine, ammonia and camphor, in asthenic cases with a tendency to a typhoid state. The *tincture of the perchloride of iron*, in doses of ten to thirty minims, *three, four, or five times a day*, in water; infusion of quassia, or calumba, is now also a remedy much in use, and it may be alternated with the *syrup of the phosphate of iron*, in doses of a teaspoonful three or four times a day. In cases where the system is obviously gouty or rheumatic, and where the joints are affected, colchicum with saline diaphoretics are the most efficient remedial agents.

Local applications are potent for good or evil, and must therefore be used with great caution. The effects seen on the skin do not constitute the whole disease; and if the development of these processes on the cutaneous tissue is imprudently interfered with, there is imminent danger to internal organs. To check the advance and prevent the encroachment upon new territory, rather than to subdue it if already in possession, ought to be the sole aim of local treatment; and to mitigate the local pain and uneasiness. Bland mucilage, such as that of viscid linseed tea, from which light muslin cloths have been steeped and spread over the inflamed surface, sometimes affords relief. Dry flour, or rye-meal, frequently dusted from a dredge-box over the erysipelatous patches, are soothing applications. A lotion of nitrate of silver painted daily over the affected parts sometimes gives great relief (one scruple of the nitrate to an ounce of water, to which ten drops of dilute nitric acid are added). Dilute nitric acid should, at the same time time be given internally, if typhoid symptoms predominate, as in the following formula:

R. Acid. Nitrici dil., ʒj; Syrup. Zingib., ʒss.; Aquæ, ʒvss.; *misce*. A tablespoonful every four hours.

To arrest the spread of the process over sound skin, nitrate of silver in very strong solution, or tincture of iodine, are efficient agents.

A line of circumvallation is to be painted round the erysipelatous part, so as completely to inclose it. The nitrate of silver should either be employed in the solid stick, or as proposed by Higginbottom, in solution of eight scruples of the nitrate with twelve drops of nitric acid in a fluid ounce of water. Dr. Wood has practised with success, and recommends the use of tincture of iodine.

[Dr. Addinell Hewson reports (*Trans. of the College of Physicians of Philadelphia*, 1867), that in extensive trials of the local use of the sulphite of soda, in solution of ten grains to one ounce of water, he has had most decided and prompt results. He has never seen it fail to arrest the progress of the disease when thoroughly applied before the deep planes of cellular tissue had been invaded. Lint soaked in the solution is thoroughly applied not only over the affected surface, but to a considerable distance beyond it, and covered with oiled silk to hinder evaporation. In the first twenty-four hours the discolored surface is sensibly bleached, and in forty-eight hours all traces of the disease have disappeared. He had thus cured twenty-seven cases of erysipelas, seven of which were idiopathic.]

Long and deep incisions into the inflamed textures are sometimes demanded. This is more especially the case if there be tension of fibrous tissue, such as the subcutaneous fasciæ; and erysipelas of the head is frequently greatly alleviated by repeated innumerable minute punctures, made by the point of a lancet all over the parts of the face and scalp which are affected.

THE PLAGUE.

LATIN, *Pestilentia*; FRENCH, *Peste*; GERMAN, *Pest*; ITALIAN, *Peste*.

Definition.—*A specific malignant fever which has prevailed at different times and places epidemically; attended with an eruption of a complex nature, composed of buboes or swellings of the inguinal or other lymphatic glands, and occasionally with carbuncles, pustules, spots, and petechiæ of various colors, and distributed in different parts of the body.*

Pathology and History.—Modern medicine restricts the term "plague" to a disease of dreadful severity, and of a peculiar character, which appears to have been first recognized in Egypt and in the neighboring countries. It is impossible to determine the time when the plague first appeared in Egypt. The remotest period to which we can distinctly trace it is when spreading into other countries, as the plague of Constantinople, which broke out in 544, when Justinian was emperor. This is the first time that the disease, from its course and symptoms, can with certainty be recognized as the plague of more modern times. The symptoms were shivering and fever, at first so slight as to alarm neither the physician nor the patient; but the same day, the next day, or the day after, there appeared swellings of the parotid, axillary, or inguinal glands, with carbuncles, and sometimes gangrene; and from the more usually diseased state of the glands it was called "*pestis inguinaria*."

The disease from that period has prevailed at short intervals in

various parts of Europe as late as the seventeenth century. Sir Gilbert Blane has calculated there were no less than forty-five epidemics of plague in the seventeenth century. Fourteen of these occurred in Holland, imported, it is supposed, by the Dutch engaged in the Levant trade, about the year 1612; and twelve in England, imported, as has been supposed, from Holland. The last epidemic of plague which prevailed in both of these countries was in 1665, the year before the memorable fire of London. This epidemic was termed the "Great Plague," and spread "with such intolerable infection" that 7165 persons are said to have died in one week, while in one year no less than 68,526 died in the city of London and its suburbs alone—an immense mortality, considering the then comparatively small amount of population.

The plague is still occasionally epidemic in Egypt, and sometimes prevails on the Barbary, Arabian, and Syrian coasts, and also at Constantinople; but it has been rarely seen out of the Turkish dominions since the seventeenth century. Nevertheless, it broke out at Copenhagen, in 1712, at Marseilles in 1720, and at Moscow in 1771. In the present century it has appeared at some of the Russian ports in the Black Sea. In 1813 it broke out at Malta and at Gozo, when the number of victims was estimated at between 4000 and 5000 (BURRELL). It subsequently broke out at Noja, in Calabria, in 1816; at Corfu in 1818; it appeared at Gussemburg, in Silesia, in 1819; and, lastly, in 1828-29, it devastated the ranks of the Russian army in Bulgaria; and there is reason to believe that at Odessa, towards the end of the recent Russian war, there were cases of a malignant fever, with buboes and swellings in the glands of the groin and axilla, which policy prevented calling plague.

It is believed that in this disease, as in others of this order, a specific poison, after a given period of latency, produces certain specific actions, which are either preceded, accompanied, or followed by fever. The more specific actions of the poison are the induction of a state very similar to that of typhus fever, as seen in this country; also a singular enlargement of the heart, the liver, or the spleen. But the most constant action of the poison is on the lymphatic system generally, as in typhus fever—the cervical, inguinal, axillary, and mesenteric glands being for the most part found enlarged or otherwise inflamed, and thus giving rise to the characteristic bubo. The areolar tissue appears to be often the seat of a specific action of the poison, in the form of carbuncles: every organ and tissue of the body is likewise covered with petechiæ, and often the seat of hemorrhagic effusion.

The extreme danger believed to attend posthumous examinations, and the prejudices of the Mohammedans, long prevented our acquiring any satisfactory data respecting the pathological phenomena of the plague; but a commission appointed by Mohammed Ali in 1834-35, and consisting of Clot Bey, Gaetani Bey, Lachesi, and subsequently of Bulard, examined the bodies of sixty-eight persons, who died of the plague, and the following is a summary of their results:

On removing the cranium, the sinuses were found filled with black blood, the arachnoid veins greatly injected, and the arachnoid cav-

ity often infiltrated with serum, and occasionally with a trifling effusion of black blood. The substance of the brain was generally less consistent than in health, and sprinkled with more bloody spots than usual. The bronchial membrane appeared sensibly inflamed, although during life the patient had presented no catarrhal symptoms. The pericardium frequently contained a reddish serosity. The serous membrane covering the heart and pericardium was often extensively affected with petechiæ. The heart, distended with blood, was almost always enlarged from a third to a half greater than its natural size, its tissues being often pale and sometimes softened.

In acute cases the stomach and small intestines were softened, and presented similar petechial appearances.

The liver was almost always larger than natural, and loaded with blood, while petechial spots were often seen at its surface. The gall-bladder was the seat of petechiæ, and in two cases blood was effused into the submucous areolar tissue.

The spleen was always twice its natural size, or even more, but was rarely the seat of hemorrhagic effusion. It was softened, and deep in color.

The kidneys were often found immersed in a hemorrhagic effusion into the surrounding tissue. They were loaded with blood, and the pelvis filled with clots. The ureters occasionally contained blood, and sometimes the lumbar glands were so enlarged as to press upon them, and to account for the suppression of urine. The bladder occasionally presented petechiæ, and occasionally the urine was mixed with blood.

Every dissection showed that buboes, wherever seated, always resulted from enlarged lymphatic ganglia, varying in size from an almond to a goose's egg. The least altered were hard and injected. In a more advanced stage some of these glands were without any change of color, and others again as richly colored as lees of wine, and either wholly or partially softened or putrescent. Sometimes these glands became agglomerated, forming masses which weighed two pounds or more, and around these agglomerations a hemorrhagic effusion extended into the areolar tissue. The cervical glands often became so enlarged as to form a sort of chaplet, united with those of the axilla and of the mediastinum. The axillary glands, again, communicated with the cervical, and with those which surrounded the bronchi. Those in the groin connected themselves in the same manner with those of the abdomen, and these might be traced without interruption through the crural arch into the pelvis and along the vertebral column. It was especially among these latter that sanguineous effusion was found in the subperitoneal tissue. The mesenteric glands were often so numerous that the whole of the mesentery seemed covered with them, but they seldom exceeded an almond in size.

In the Mediterranean cities, where plague epidemics have prevailed, it is of importance pathologically to remember that epidemics of "anthrax," "carbuncle," "phlegmon," "boil," or "pustule," are not uncommon. The disease usually shows itself in the form

of tumors at the lower part of the neck, between the shoulders, or in the loins. These vary in size from that of a pigeon's egg to a circumference of eight inches. They are preceded by shivering, headache, prostration, sickness, occasional vomiting, and sleeplessness, succeeded by fever, the appearance of the carbuncle, and typhoid symptoms. In some years the disease is severe, and yields a high mortality. The subjects of such disease are generally persons of sickly appearance, of gross habits of life, and who live in damp, filthy localities, where the plague formerly committed its ravages. It is extremely probable that these diseases are cognate to the true plague—that, having been born together, the source of the true plague is always in existence where these diseases prevail, requiring only the requisite unfavorable sanitary conditions to call forth the epidemic pestilence (see *Report on the Sanitary Condition of Mediterranean Stations*, by Capt. Galton and Dr. Sutherland, pp. 88, 89).

Symptoms.—The poison of the plague produces those disordered functions of the great nervous centres which constitute the phenomena of fever, either of a low or of an active character, and sometimes so severe as to destroy the patient within one or two days, and before any secondary lesions are set up. "At Aleppo," Dr. Russel says, "in the most destructive forms of the plague the vital principle seems to be suddenly, as it were, extinguished, or enfeebled to a degree capable only for a short time of resisting the violence of the disease; and the form of the plague beyond all others most destructive exists without its characteristic eruptions, or other external marks considered pestilential. These cases perished sometimes within twenty-four hours."

The manner in which the disease commences varies, but generally it is preceded for a greater or less length of time by "lassitude, loss of strength, general uneasiness, and mental anxiety, to which shivering, headache, vertigo, and vomiting soon succeed; then appear the general and local phenomena, and among them the characteristic bubo, carbuncles, and petechiæ, preceded or followed by delirium or coma, too often terminating in death."

The buboes of plague seldom mature till the fever is on the decline, which rarely happens till the eighth or ninth day; nor are they generally ripe for opening till between the fifteenth and twenty-seventh day. In general, suppuration has not been so frequent as resolution, and never were the buboes seen to be gangrenous. Aubert considers the bubo as of good augury for the patient, and its suppuration as the sign of his recovery.

The carbuncle is by no means of constant occurrence, Dr. Russel having found it only in 490 cases out of 2700. It appears more commonly in the middle or towards the decline of the disease. Hardly any external part is free from them, not even the penis; and in one instance a carbuncle formed in the throat, which was fatal. They occur more particularly on the limbs, and more especially on the legs. In some cases they form on the cheek or lips, and, by the tumefaction they cause, give to the face a hideous aspect; in others the whole of one side of the jaw has been laid bare; while in others they have formed on the eyebrow and on the eyelid, and partly

destroyed the eye. Clot Bey, however, observed they never formed on the scalp, the palms of the hands, or on the soles of the feet.

There are three different varieties of carbuncle, and all commence in the same way, or by a small red pimple, which increases, and in the centre of which a vesicle forms, containing first a yellow and afterwards a blackish serum. In the most benign the vesicle bursts, and dries up in three or four days from its first formation, the epidermis alone having been infected. The second variety involves the whole thickness of the skin, as well as portions of the cellular tissue, which is moderately tumefied, and surrounded by a dark-red areola. The gangrene in this form is circumscribed, and there results an eschar from one to two inches in diameter, which is detached by suppuration, leaving an ulcer with a sharp perpendicular edge. In the severe forms the redness and tumefaction cover a large space, and the gangrene rapidly involves the skin, the cellular tissue, and sometimes even the bones. It has been observed that the malignity of the carbuncle is in the direct ratio of the severity of the disease, but the mere existence of carbuncle is not of unfavorable augury. Their number is very various, sometimes only one, at others ten or twelve. When there are several, they often form in succession. These tumors are often very painful; and Aubert mentions one, seated on the back of an Arab soldier, four inches in diameter.

Petechiæ are observed in some seasons and not in others. They present different shades of color, according to the intensity of the disease—rose color, violet color, or black. Aubert considered their appearance an almost certain sign of death. The duration of the disease is from a few hours to fifteen, twenty, thirty, or even more days.

Diagnosis.—Clot Bey says the diseases which most resemble the plague are typhus fever, severe forms of paludal fever, apoplexy, dysentery, parotitis, and scrofulous or syphilitic affections associated with febrile symptoms of a typhoid type.

Cause.—The plague, and the specific poison which it generates, seem to have a very limited geographical range. Clot Bey, indeed, considers it to be endemic along the whole of the eastern and southern coasts of the Mediterranean, the principal centres of propagation being Egypt, Syria, and Constantinople. But most authors are agreed that Egypt is the great focus of the plague, whence it may be propagated under circumstances of overcrowding, filth, dampness, and organic decomposition. It seems determined also that the disease is often circumscribed within a very small space of country. Volney states that in Egypt the plague never commences in the interior, but always appears first on the coast at Alexandria, passes from Alexandria to Rosetta, and from Rosetta to Cairo.

All that we can safely affirm of the poison of the plague is, that it is at all times endemic in Egypt, along with the cognate diseases of "carbuncle," "anthrax," or "boil," already referred to, and every five or six years it becomes epidemic. It also appears to be, to a certain extent, influenced by season, not spreading in any very

sensible degree till December, and attaining its greatest height in June, when it rapidly declines.

The period of the year, however, at which the plague prevails differs in some degree in different countries; but the total duration of the disease in any country to which it is not native appears to be inconsiderable, unless kept up by a fresh importation. At Aleppo it lasted from 1760 to 1762, a period of three years. But in Malta, Marseilles, and in the western parts of Europe, it has generally subsided in about twelve months.

In selecting its victims, this poison follows the law of most other morbid poisons, attacking the poor rather than the rich,—women rather than men,—patients laboring under disease rather than healthy individuals,—persons constitutionally feeble rather than the robust, and those addicted to intemperance, or other excesses, rather than those who more strictly observe the precepts of Mohammed. As to races—the Arab suffers more than the Negro, the Negro than the Turk, and, in Egypt, the Turk more than the European.

Modes of Propagation.—The belief that the plague is capable of being communicated is so general that it still continues to be the terror of Europe, and the ports of every nation are closed against a vessel supposed to have the plague on board. The facts by which this precaution is warranted are extremely striking; for every time the plague has appeared in Christian Europe, the arrival of a ship has been an invariable antecedent on board of which one or more persons have died of the plague. The disease, also, invariably broke out at the port or town at which such vessel arrived; and if proper precautions were not taken, it spread into the interior of the country.

It is known that the antecedent arrival of a vessel having the plague on board, at each of the three ports of Marseilles, Messina, and Malta, and the breaking out of the disease in all those places shortly afterwards, is so remarkable that it can be only explained by admitting, in these instances, the connection of cause and effect. Moreover, the fact of the plague having originated in the preceding instances from imported cases of the disease, and not from any local influence, is demonstrated by the exemption of large bodies of persons "shut up" in the very heart of the pestilence. Thus, in the plague at Marseilles, the large nunnery of Les Dames de la Visitation Sainte Marie "shut up;" and although there was an infirmary on one side for those ill of the disease, and a burying-ground on the other for those who died of it, yet all the inmates of the nunnery escaped. The Hôpital de la Charité of the same city, a sort of poor-house, making up about 300 beds, "shut up," and escaped with complete impunity; but being converted into an infirmary for the plague patients, 200 of the poor, left in attendance, all died of the malady.

Another class of facts demonstrative of the communicable nature of the plague is the great number of persons attending on, or in communication with, the sick, who die from this disease. The French army, on first taking possession of Egypt, lost no less than eighty medical officers by the plague—an immense proportion compared

with the loss of the army generally. In the English army only one in forty-eight of the military died of the plague, while one-half of the medical officers died. Some few persons also have ventured voluntarily to inoculate themselves with plague-matter, and these have, with hardly an exception, fallen victims to their rash experiments.

Dr. Russel states that at Aleppo he met with twenty-eight cases of re-infection, or 1 in 157; and Clot Bey states that he and his colleagues saw many individuals perish of plague in 1834-35 who had formerly survived an attack of the disease.

Treatment.—In the treatment of the plague neither the practice of the French nor English medical officers serving in Egypt has led to any happy result; and it is to be regretted that recent experience has not in any degree advanced the successful treatment of the plague. "In the beginning of the epidemic," says Clot Bey, "when the morbid cause acts with a rapidity so great that some hours are sufficient to compromise the life of the patient, every treatment, even the most energetic, is powerless to arrest the course of the disease. When, however, the intensity of the disease abates, we may hope for the recovery of the patient." Looking, however, to the pathology of the disease, and regarding it as a form of malignant *typhus fever*, the principles of general treatment ought to be similar to those laid down in the account of that disease.

We have no sufficient evidence to prove that plague may be carried beyond those geographical limits where it or the cognate diseases already noticed are epidemic. Quarantine establishments to prevent the transmission of such epidemic diseases are now therefore unwarrantable nuisances, and vexatious interruptions to mercantile enterprise. I am informed by a medical friend who visited Malta in 1861, that a curious instance of the wavering nature of opinion regarding the efficacy of quarantine was afforded during the last Benghazi plague. The Maltese—the most sensitive people formerly on the subject—absolutely declined to put any quarantine on arrivals from Benghazi, and trade went on as usual until the Austrians intimated that, unless Malta put Benghazi in quarantine, Trieste would put Malta in quarantine; and the poor merchants were obliged to submit. There is little risk of plague now, because of the great improvements which have taken place; and it is to be hoped that as sanitary measures are developed the barbarism of quarantine will entirely disappear; except, perhaps, where the intelligence of the people does not go beyond that of the Governor of Eupatoria, who requested that the allied armies of France and England might go into quarantine when they landed in the Crimea in 1854! (KINGLAKE.)

SECTION II.—THE CONTINUED FEVERS.

Fevers have been classified according to various theories; and much has been written on the subject. In the previous section those diseases have been described in which an eruption on the skin especially challenges attention, and with the appearance and development

of which more or less fever is associated. They are described by most systematic writers as a separate class, under the name of "The Eruptive Fevers."

The "Continued Fevers," now about to be described, were at one time all recognized under the single name of "Common Continued Fever," of which it was believed there were several varieties. But since about the year 1840 specific differences have been gradually becoming more and more obvious, so that now at least FOUR DISTINCT FEVERS can be recognized, allied by certain common characters, and not less distinctly separable by peculiar and distinctive marks. The plurality of continued fevers must now therefore be generally admitted.

The four fevers about to be described have been mixed up together in almost every epidemic, in various proportions, so that each epidemic of fever has held a peculiar character, according to the nature of the dominant disease which was mainly prevalent.

The four forms of Continued Fever are now named respectively as follows: (1.) Typhus fever; (2.) Typhoid, Enteric, or Intestinal Fever; (3.) Relapsing fever; (4.) Febricula.

Much has been spoken and written about the identity and non-identity of *typhus* and *typhoid* fevers; and in the first edition of this work I stated my belief that these two forms of continued fever were identical in their nature—*i. e.*, were varieties merely of a fever which resulted from one and the same specific poison. I entertained this belief partly because I had been taught as a student so to believe, and partly because I considered that the evidence then existing on the subject, and with which I was acquainted, did not fully justify any other conclusion. This belief I recanted in the second edition, for proofs of numerous and remarkable differences of a specific kind between *typhus* and *typhoid* fever have been slowly but surely accumulating since the beginning of the present century. The dissections, by Prost, of Parisian fever patients in 1804, may be said to have laid the foundations of our knowledge, and to have turned the attention of pathologists in the direction which has led to such definite results. In more than 150 dissections he always found "inflammation," with or without ulceration of the mucous membrane of the intestines. Petit, Serres, Pommer, and Bretonneau followed up the investigations of Prost; but the celebrated treatise of Louis, in 1829, was the first to give a complete and connected view of symptoms as well as of post-mortem lesions in the fever common in Paris; and although Prost asserted the connection of a certain intestinal lesion with a definite series of symptoms, still it was Louis alone who described this intestinal lesion in terms sufficiently precise, and indicated with scientific exactitude the symptoms with which it is concurrent. The views of Louis were subsequently adopted by Chomel and Andral in France. In other parts of Europe, however, and especially in England, bodies of numerous fever patients were opened without finding any disease of Peyer's patches, although differences in the symptoms detailed regarding the fevers of France and England were not then so obvious. Hence arose at once two opinions, based on a post-mortem distinction,

the "anatomical sign," described by Louis. The first opinion was that this "anatomical sign" was an incidental occurrence; or, that its occurrence was in some way connected with locality, the cases of fever being everywhere considered identical. A second opinion, however, soon began to gain ground, especially when the intestinal lesion was not found by the most careful observers in some cases where it was intentionally looked for (as in the epidemic of Toulon in 1829-30). A belief now, therefore, began to gain ground that there were in fact *two diseases*, which were indifferently named *typhus* and *typhoid fever*—that one prevailed only at Paris, and the other in England, in Germany, and elsewhere, being also sometimes more or less mixed up with the Parisian fever, as measles may be with scarlet fever. Louis subsequently (1841) adopted this view.

In 1835 the "Académie de Médecine" formally proposed the question, "What are the analogies and the differences between the *typhus* and *typhoid* fevers?" The question excited considerable interest in France, but less so in England, where a strong bias has always prevailed towards a belief in the doctrine of a single fever—a belief entertained and taught by the most eminent observers and teachers of the day. But dissenters arose. Scotch, English, and American physicians, practically familiar with the fevers of their countries, began to visit Paris to study fever there; and they were not long in learning to recognize the chief point of difference between the two fevers. Gerhard and Pennock, of Philadelphia, in a systematic treatise, were the first to indicate (1836) these differences, it having been already determined by Jackson and Gerhard that the fever described by Louis under the name of *typhoid fever* existed in America, and presented there the same assemblage and development of symptoms, and the same post-mortem lesions, as the Parisian fever.

In 1836 M. Lombard, of Geneva, after visiting London, Edinburgh and Dublin, ultimately came to the conclusion that two different fevers had been confounded together; and Drs. Staberoh, of Berlin, and Kennedy, of Dublin, professed the same belief through the same medium—the *Dublin Journal*. During this year (1836), also, Dr. A. P. Stewart commenced his observations in the Glasgow Fever Hospital, where he continued his inquiry for two years. His attention was first especially directed to the study of fever by Dr. Peebles, who, during a long residence in Rome, had observed the maculæ of typhus in the contagious fever of Italy, and who first showed the difference between the characteristic eruption of fever and the cutaneous affection to which the name of "*petechiæ*" is given (*Edin. Med. and Surg. Journal*, 1835). He pointed out this eruption to Dr. Perry (then Physician of the Glasgow Fever Hospital), "and who," Dr. Stewart states, "was the first to maintain the complete difference of the two eruptions—namely, those of *typhus* and *typhoid fever*." Dr. Stewart subsequently went to Paris and examined the fever there. The result was a complete recognition of the existence of two fevers, and of their differences—an account of which he published in the *Edinburgh Medical and Surgical Journal* for 1840, p. 289.

In 1839 Enoch Hale published an account of the fever of Massa-

chusetts, and distinguished among them two perfectly different forms of fever, one of which agreed with the Parisian fever, while the other might be held to represent the fever described by most English writers.

[In February, 1835, Dr. Gerhard, of Philadelphia, and in June, 1835, Dr. Bartlett, indicated with great precision the difference between typhoid fevers and typhus.]

Soon after this the characters of the prevalent fevers of England were noted by Shattuck (another American pupil of Louis), who published his results in the *Boston Medical Journal*.

The appearance of these papers, and of others about this period, gave rise to an elaborate discussion of the whole question (in the pages of the *British and Foreign Medical Review*, vol. xii, p. 293); and the conclusion the reviewer arrived at seemed to favor the opinion, "that the French and English fevers were varieties, that is, different developments, of a common stock, but not specifically distinct diseases." I understand the eminent physician who wrote that review now believes in the specific distinction of the two fevers.

In America, the doctrine of a specific distinction between the two fevers has been generally adopted; as represented in the treatises of Dr. Bartlett, of Philadelphia, in 1842 and 1847, *On Typhus and Typhoid Fevers*, and *On the Fevers of the United States*; [and by Dr. Meredith Clymer, *Fevers, their Diagnosis, Pathology, and Treatment*, Philadelphia, 1846, p. 234.]

In Germany three opinions were entertained. The typhoid fever of Louis received from the Germans the name of "abdominal typhus"—thus regarding the disease as a variety of typhus fever. By some, however, it was regarded as a disease distinct from the "*typhus exanthematicus*." A third opinion also found followers—namely, that this *abdominal* typhus was the only form of continued fever—the result of a limited and narrow field of investigation.

Up till 1846 opinions were thus divided, crude, and in not a few instances quite unformed. Relapsing Fever was distinguished by some, but not by all; and all other forms of continued fever were considered in this country as identical. Under those circumstances the inquiry was taken up in 1846 by Dr. Jenner, then Professor of Pathological Anatomy in University College, and worked out by him systematically in the London Fever Hospital. There he patiently accumulated case after case of fever, until he had nearly 2000 accurate reports before him. From these he separated all cases of relapsing fever, and then instituted a rigorous comparison of the remaining cases. He selected the fatal cases which had been examined after death, and the diagnosis of which had been confirmed. He found that he had 66 such cases and post-mortem examinations. Of these 66 cases, 23 had the intestinal and mesenteric lesion—the "anatomical sign" (according to Louis) of typhoid fever; and 43 cases were without this appearance. The question then remained for solution; namely—Did these 43 cases (in which the intestinal lesion was not present) differ so much in symptoms and

post-mortem appearances from the other cases (in which the "anatomical sign" referred to was present) as to render it impossible to suppose that they were cases of the same disease? Or,—contrary to the opinion of Louis,—Were the symptoms of the two sets of cases so similar as to lead to the belief that the presence or absence of the intestinal lesion (the "anatomical sign") was a matter of little consequence?

On comparing these two groups of cases, Dr. Jenner found that while the symptoms and post-mortem appearances of the 23 cases were exactly the same as those described by Louis, the symptoms, course, and post-mortem appearances of the remaining 43 cases were entirely different—so different, indeed, as to render their separation from the other cases a matter of absolute necessity, if accuracy was to be maintained in the description of these diseases, or certainty arrived at in their treatment.

Causation, as a ground of distinction between the two fevers, is a condition upon which much stress has been laid by Dr. Jenner, and subsequently by Dr. Murchison. Dr. Jenner was the first to argue that the material media by which the two fevers are propagated are specific and different from each other, according as they are generated by the bodies of those affected with the one or the other form of fever. This argument he based upon the circumstance, that because *certain local foci* sent *typhoid* cases to the hospitals, and *certain other local foci* sent *typhus* cases there, he inferred that different specific causes existed in each focus. Dr. Murchison has also clearly stated the evidence of many other observers, which goes to prove that the two fevers have no community of origin (*Continued Fevers of Great Britain*, p. 588).

[Dr. Southey Warter (*St. Bartholomew's Hospital Reports*, 1866) considers the thermometric differences between typhus and typhoid fever so great as to completely settle any doubts as to their being separate diseases.]

This brief history of the progress of our knowledge regarding typhus and typhoid fevers has been mainly condensed from an erudite and most interesting monograph on "The Diagnosis of Fevers," by Dr. Parkes, which appeared in the *Medico-Chirurgical Review* for July, 1851—a contribution of not less importance to science than the original investigations of those whose labors it records; for it connected the scattered observations together, and showed at once the practical value of the discovery that had been so gradually maturing, as it did, to bring conviction to the minds of those not fully conversant with the literature of the subject, and with what had actually been achieved in different parts of the world. To Dr. Parkes, the clear, elaborate, and careful analysis he made was a labor of love—justly believing, as he does, that no subject is so important as an accuracy of diagnosis. It is the foundation of therapeutics; and he who clearly indicates how a disease can be recognized is fellow-laborer to him who points out how the disease may be cured or prevented.

This brief history teaches us how slow is the progress of discovery. The greatest discoveries have been rarely due to any single individual; but gradually, slowly, and surely the Light of Science dawns upon the world. It was so with the discovery of the Circulation of the Blood. It was so with the discovery of the Protective Influence of Vaccination. It was so with the discovery of the Powers of Steam, and the development of the steam engine to its present condition of perfection.

Since 1851 proofs of differences between the two fevers have been still accumulating in many different directions. They especially result from the observations of Dr. William Budd, of Clifton, near Bristol; of Dr. Murchison, of the London Fever Hospital; of Professor Wunderlich, of Leipsic; and of Von. W. Greisinger, of Zurich. The observations of these two latter physicians are especially valuable, as showing the ranges of temperature in the two fevers to be distinctive of two diseases. I must here also mention the excellent lectures given by Dr. Peacock, of St. Thomas's Hospital, in 1855, and published in the *Medical Times* of 1856, "On the Varieties of Continued Fevers and their Discrimination," as influential in forwarding these modern views, for they appeared at a time when the specific distinctions between typhus and typhoid fevers were less generally admitted than at present. Thus the evidence has slowly but surely accumulated; and when the whole subject has been re-examined in all its relations, the conclusion irresistibly forces itself on the understanding, that a belief in the identity of *typhus* and *typhoid* fevers is no longer tenable.

In common with many, I had, as a student, been taught to recognize the striking similarity between the two fevers, in outward aspect, in many respects; and therefore I was unduly biassed by the *resemblances*, rather than led to give sufficient importance to the numerous and remarkable *differences* between them which are now to be described.

With regard to their most prominent points of resemblance and difference, it may be shortly stated here, that all the points in which the two fevers agree are common to them and many other diseases, and therefore are of no value as *indicia* of a species. On the other hand, the points in which they differ are all of a very special nature. The points in which they agree may all be summed up in the phrase "typhoid symptoms"—a set of symptoms which are met with in a great variety of diseases, and therefore are of no specific value in the question at issue. These so-called typhoid symptoms (represented by the phenomena of stupor, low delirium, general prostration, *subsultus tendinum*, a dry and incrustated mouth, deafness) occur not only during the course of *typhus* and *typhoid* fevers, but are also found to occur and to group themselves in a similar manner in *pyæmia*, *uræmia*, some forms of *pneumonia*, and in many cases of *acute tubercle* (W. BUDD).

ENTERIC FEVER—SYN., TYPHOID FEVER.

LATIN, *Febris enterica*; FRENCH, *Fèvre typhoïde*; GERMAN, *Abdominal-typhus*—Syn., *Ileo-typhus*; ITALIAN, *Tifo enterico*.

Definition.—A continued fever associated with an eruption on the skin of rose-colored spots, chiefly on the abdomen, appearing generally from the eighth to the twelfth day, occurring in crops, each spot continuing visible about three days. Languor and feebleness are prominent from the first, attended by headache, abdominal pains, and (early) by spontaneous diarrhœa. With the advance of the disease the diarrhœa increases, the discharges being for the most part liquid, copious, of a bright yellow color, devoid of mucus, occasionally containing altered blood; in reaction alkaline, and containing a large proportion of soluble salts and some albumen. The disease may terminate favorably by a gradual restoration to health during the fourth week. The average duration of the fever is about twenty-three days. Death in the majority of fatal cases occurs towards the end of the third week. There are symptoms also associated with the characteristic lesion of this form of fever—namely, fulness, resonance, and tenderness of the abdomen; more or less tympanitis, with entire effacement of the natural lineaments of the belly; gurgling in the iliac fossæ; increased splenic dulness. The specific lesions are enlargement of the mesenteric glands, with deposit in the glands of Peyer and in the minute solitary glands of the small intestine.

Pathology and Symptoms.—Typhoid fever begins gradually—often, indeed, so very insidiously that its commencement is not always able to be fixed.

This form of continued fever is described under a great variety of names, by various writers, such as *typhus mitior*; *nervous fever*; *abdominal typhus*; *common continued fever*; *entero-mesenteric fever*; *donthenteritis*; *follicular enteritis*; *bilious fever*.

The fever may be ushered in with rigors, chilliness, or profuse diarrhœa; and amongst the early symptoms, the most characteristic are the abdominal pains and diarrhœa, which continue to increase. [There is early muscular debility, shown by the staggering walk, and, subsequently, by dorsal decubitus.] The countenance indicates anxiety, [and has a distinctive besotted expression]; the mind continues clear, [but intelligence soon becomes weakened, and questions often have to be repeated before understood and answered; this partly depends on dulness of hearing, with ringing in the ears, which are very constant]; and delirium, when present, is generally active. [Frontal headache is a constant initial symptom, and often insomnia.] The patients are vivacious, and disposed to leave their beds. The conjunctivæ are pale, the pupils dilated, the cheeks somewhat flushed, and slight, though sometimes excessive, epistaxis not seldom occurs, at repeated intervals, during the first week. The belly enlarges, as in mesenteric disease, and is resonant on percussion. Gurgling on firm pressure may commonly be detected in the right iliac fossa, and there is often tenderness in the same situation, [with pain, on pressure, around the umbilicus.] From the seventh

to the fourteenth day the characteristic eruption appears. As a rule, the flushing of the face is more marked towards evening; but the complexion does not get muddy, as in typhus, and the flush of the cheeks is bright and pinkish—not dark red—and is often circumscribed, and then strongly contrasts with the surrounding pale skin. During the third week the abdomen becomes more distended; the diarrhoea increases, the stools often amounting to *five, six, or even eight and ten* a day. They are liquid, pale brownish-yellow, with flocculi of an opaque whitish-yellow color floating through them like coarse bran, and as the patient loses strength they are passed involuntarily. Pain is rarely complained of unless perforation of the gut occurs; and hemorrhage from the bowel is an occasional symptom during the third or fourth week. The frequency of the pulse often varies much from day to day, without any appreciable coincident alteration in the general or local symptoms. It is generally soft. [The tongue is early overspread with a whitish-yellow, or brown fur,] then red and fissured, but ultimately becomes dry and covered with a pale-brown fur, [or, coated at the base and in the centre, with deep red glazed edges and tip.] Thirst is urgent and constant; the secretions of the mouth thick and glutinous. As the mouth dries, the whole mucous membrane assumes a uniform red color; the lips crack, and the teeth look bright from the dried layer of mucus covering them. There is complete anorexia, with often vomiting of bitter and greenish matters. The splenic dulness is generally increased, [sometimes extending below the margin of the ribs.] Pulmonic complication is not uncommon.

[Slight cough frequently exists from the outset, with expectoration of viscid greenish sputa, and quickened respiration. Sibilant and sonorous rhonchi are heard, unequally diffused, on both sides of the chest, but usually more marked inferiorly and posteriorly. We have several times met with general bronchitis of both lungs in typhoid fever without a single objective symptom, and made out only by physical exploration. The bronchitis is of a congestive form, as well as the pneumonic complication, which latter is infrequent except in the later stages of the disease, when it is hypostatic, rarely accompanied by rational symptoms, and made known only by its physical signs.

In severe cases the symptoms of the nervous centres increase about the second week; the patient lies motionless on his back, or there is a tendency to slide down in the bed; he seems perfectly unconcerned, and desires to be let alone; questions, when heard or understood, are slowly and reluctantly, or petulantly, answered,—the replies being often brief and dry; the perception of surrounding objects is vague; the cast of the face is stolid; the eyes are injected and brilliant, but have a stupid expression; sleep is dozing, unrefreshing, and disturbed by vivid and startling dreams, with confusion and incoherency on awakening, or, more rarely, there is persistent wakefulness; the headache grows less or ceases; deafness is complete; the respiration is sighing and spasmodic; there is a tendency to irregular and involuntary movements of the tendons of the forearm, with carphology and twitchings of the muscles, upper lip, and nose. Delirium, when it happens at this stage, is manifested towards night, the usual period of febrile exacerbation, and is commonly tranquil, with rambling muttering, and a disposition to leave the bed and roam around, though

sometimes it is violent and loud, and occasionally hysterical. The face is swollen and dusky, and the skin over the malar bones of a livid red, and there is often general capillary sluggishness of the surface of the body. The pulse is soft, rapid, and, sometimes, irregular; the duration and intensity of the first sound of the heart will, in many cases, be found lessened, and may become quite extinct.

Towards the end of the second week, or the beginning of the third, there is either a gradual, though marked abatement in the symptoms, or they suddenly and quickly worsen. If the attack is to end in recovery, the temperature of the body lessens, chiefly in the morning, and the skin becomes moist and soft; the tongue cleans, and the buccal secretions return; the expression of the face begins to look more natural, and the pulse is slower and steadier. Or, if the duration of the disease is to be prolonged, a decided increase in the severity of the symptoms will take place, and new ones will be added. The tongue becomes drier, browner, fissured, and trembling; sometimes it is of a bright red color and smooth, as if covered with a coat of varnish. The mouth and teeth are crusted with dark sordes; there is great difficulty, or even inability, to protrude the tongue, or to swallow, which may be due to paralysis of the muscles of deglutition, but more often to the half-dried mucosities gathered about the base of the tongue. The nostrils become blocked with dried mucus or blood, and the breathing has a peculiar whistling sound. The pulse is quick and irregular, reaching 120 beats or more; meteorism is excessive, and diarrhœa is profuse and often, the stools passing involuntarily; there is retention or suppression of urine, or, though rarely, incontinence; and hemorrhages may happen from the nose, bowels, or vagina. Bronchitis becomes more general and intense, and pneumonic complications set in; at this time uræmic coma may come on, and petechiæ appear. The heat of the body is acrid; a peculiar odor is, sometimes, exhaled, said by some to be like that of mice; sloughs are common on those parts of the body which have been exposed to pressure, as over the sacrum, heels, scapulæ, trochanters, &c., or gangrene may attack blistered surfaces, or leech-bites, or parts of the skin where sinapisms have been applied. Sometimes spontaneous sphacelation takes place; Dr. Grisolle has seen gangrene of the integuments of the thigh, scrotum, foot, and lower lip come on in the course of typhoid fever without any obvious cause.

The duration of convalescence is generally proportionate to the sharpness of the attack; when this has been severe and protracted, and the prostration great, strength is slowly gained, and recovery is very gradual. Emaciation is often excessive at the beginning of convalescence. Painful œdema of the lower extremities, rarely extending to the upper limbs, and face, and loss of the hair of the head are frequent. In many cases the hearing remains dull for some time, particularly where there has been a purulent discharge from the meatus. Convalescence may be suddenly arrested by symptoms of gastric disorder, the digestion becoming difficult, the skin hot, and the pulse quick, arising from some irregularity of diet; they commonly abate after a day or two. Abscesses, eschars, erysipelas, and successive crops of boils on the trunk and extremities, often lengthen convalescence. A persistent frequency of pulse may last for some weeks. Autophagic vertigo is not uncommon after a protracted attack, and particularly where the patient has been imperfectly nourished. Paralysis, dependent on deficient innervation, both of sensation and motion, may complicate convalescence, causing blindness, deafness, paresis of the lower extremities, or loss of power in the sphincters of the rectum or bladder. Softening of the cornea has been noticed.

[**Spinal Symptoms in Typhoid Fever.**—Certain symptoms happen occasionally in the course of typhoid fever, which show more or less disturbance of the functions of the spinal cord. They have been but little noticed by systematic writers, and have generally been looked upon as accidental complications, or referred to spinal or cerebro-spinal meningitis.* They should not be confounded with the consecutive cerebral disorders already mentioned. Their recognition and appreciation are important both as regards diagnosis and prognosis. Of irregular occurrence, they are much more common at one time than at another, being more frequent when typhoid fever is epidemic, and when malarial toxæmia is a complicating element; and are most often met with in children, women, and anæmic individuals. Of 44 patients (30 males and 14 females) admitted into the wards of Dr. Tardieu, at the Laraboisière Hospital, Paris, from the 31st of July to the 23d of October, 1863, with typhoid fever, spinal symptoms were present in 26 (13 males and 13 females). The average age in 12 females was 23 years, and in 13 males 22 years. Of 31 observations collected by Fritz, 13 were between 5 and 13 years, and 18 between 17 and 40 years; of whom 11 were aged 20 years or under, 3 were between 20 and 30 years, and 4 between 30 and 40 years.

Spinal symptoms in typhoid fever may be initial, or they may be developed in the progress of the disorder. They may be transient or persistent. When prodromic, and of exceptional severity, they may be predominant and alone fix the attention of the patient, being the illness of which he complains, and for which he seeks relief. They are marked by aching pains in the lumbar, dorsal, or cervical regions, particularly in the back of the neck, radiating to the occiput, and interfering with the motions of the head and neck; shooting pains in the limbs, most often the legs; with a feeling of stiffness and numbness in the muscles, especially those of the jaw; and more or less cutaneous and muscular hyperæsthesia. These may subside on the development of the disease, or continue to the middle or end of the first week, and then cease; or they may last quite through the disorder, and even reach to convalescence. Again, but more rarely, they may appear during any stage of typhoid fever. These nervous troubles have two distinct origins: one set of phenomena is due to functional derangement of the spinal cord proper, and the other to that of the medulla oblongata. Beginning with those referable to the spinal cord proper, they may be arranged under two heads: (1.) Derangements of the sensory functions; (2.) Derangements of the motor func-

[* Whilst intercurrent nervous affections in typhoid fever, referable to the brain, cerebro-spinal system, and the sympathetic, have been fully described by writers, those due to the spinal cord alone, have either had but slight recognition, or been passed by, and have generally failed to receive any precise, or physiological interpretation. That they have been observed from time to time for many years is shown by looking over the vast bibliography of typhoid fever, and most physicians who have had large experience of this disorder cannot but have frequently met with them.

To Dr. Fritz is really due the credit of having first studied understandingly the nervous phenomena in typhoid fever, referable to the spinal cord, and clearly establishing their true pathogeny. His observations, made in the wards of Dr. Barthéz, at the St. Eugénie Hospital, and in those of Dr. Tardieu, at the Laraboisière Hospital, Paris, are very clearly detailed, together with the most prominent ones on the same subject already published, as well as those sent to him by Prof. Shutzenberger, of Strasbourg, and Dr. Benckard, of Kaisersberg, in his admirable *Etude Clinique sur divers Symptômes Spinaux observés dans la Fièvre Typhoïde*, Paris, 1864. The writer has freely used this valuable essay.—EDITOR.]

tions. Derangements of the sensory functions are of three kinds: (a) Exaltation of function,—hyperæsthesia and spontaneous pains; (b) Perversion of function,—abnormal sensation of cold or heat, prickings and formication in the extremities and along the spine; much more rare than the first variety, and, usually, when present, associated with it. (c) Diminution or abolition of function,—the several degrees of analgesia and anæsthesia, cutaneous and muscular.

Hyperæsthesia may be limited to the skin, or to the muscles, or both may be affected. Cutaneous hyperæsthesia may extend over a considerable portion of the body. Its site is often the skin of the abdomen, or of the extremities, or conjointly, the lightest pressure, or the merest touch being intolerable; when it is of less degree it may be provoked by gently pinching a fold of integument, or passing the finger over the internal face of the tibia, about the malleoli, or the condyles of the femur. Next in frequency we have increased sensibility over the spinous processes, sometimes reaching from the atlas to the sacrum, and sometimes limited to a single apophysis, and induced by pressure. There may be severe aching pains in the muscles, generally of the lower limbs; or rachialgia, radiating to different parts of the body, and increased by any movement; violent pains in the chest and waist; and neuralgia, which is commonly symmetrical. Both cutaneous and muscular hyperæsthesia may be chiefly complained of toward evening, when the body-temperature rises. Its progress is generally regularly ascending, and it disappears in inverse order.

Dr. Robert Law, of Dublin, thus graphically describes the spinal symptoms which were observed in the Famine Fever of 1848: "The most common and loudest complaints of our patients was an aggravation of what the subject of fever generally describes as pains in the bones, but which really means pains in the course of the spinal nerves. While in former fevers this complaint was seldom more than that of a contused or bruised feel, or of such a sense of discomfort or fidgetty restlessness as the French so significantly express by the term *malaise*, here the individual in many cases seemed to suffer as intensely as in the severest cases of acute rheumatism. These pains were more or less general in different patients. In some they affected the back of the head and neck; in some they only ran down the legs; while in others they spread themselves through the whole body, and embracing the sides, imparted the sensation of painful constriction. The nape of the neck and across the loins were the points to which the patients most frequently referred their pain." (*Dublin Quarterly Journal of Medical Science*, Nov., 1849.)

Derangements of the motor functions are: numbness of the extremities, paraplegia, partial paralysis of the respiratory muscles, retention of urine, paralysis of the sphincters, spasm, or irregular contraction of the muscles of respiration or of the extremities, and muscular rigidity, particularly of the muscles of the neck and limbs. The special group of nervous symptoms originating in the medulla oblongata, are: extreme breathlessness, not due to any morbid condition of the organs of respiration, spasm of the pharynx and larynx, convulsive cough, aphonia, glossoplegia, spasmodic or rhythmic contraction of the sterno-cleido-mastoid and trapezius muscles, and paralysis of the pharynx.

When a patient with typhoid fever has died after or during the occurrence of the spinal symptoms just described, no appreciable material lesion has been found in the cord or its membranes. In a very limited number of cases can these symptoms be referred with any strictness to a congestion of the cord (FRITZ).

When the spinal symptoms of typhoid fever have been very marked and

severe, and especially when they have been associated with cerebral disorders, they have sometimes led to an error of diagnosis; and there is no doubt that there is a good deal of analogy between them and many of the phenomena of cerebro-spinal meningitis, and at times, it must be owned, a differential diagnosis is difficult. As a general rule a mistake can be easily avoided by contrasting the integrity of some of the functions of the cord with the great perversion of others; by the mobility of the symptoms and their irregular succession; by the presence of the distinctive features of the idiopathic fever; the difference in the invasion, sudden in the one, gradual in the other; and the expression of face, anxious and betokening suffering in cerebro-spinal meningitis, besotted in typhoid fever.

When the symptoms indicate that their origin is in the medulla oblongata, the prognosis must be guarded. Breathlessness, without pulmonary complication, is almost always a token of a speedy fatal ending.]

In cases that recover, a remarkable fatuity remains behind long after recovery; and there appears to be some diminution of intellectual power for some time after convalescence is restored. Dr. Jenner has seen many cases in which childishness of mind remained for more than a month after apparent restoration to health. The patient generally wakes up, as it were, from the fever a complete imbecile. The whole man is changed. He seems to have renewed his youth. Childhood and infancy return, and the greatest care is necessary to prevent untoward events. NO MAN CAN BE CONSIDERED AS FIT FOR WORK, OR FOR GENERAL MILITARY SERVICE, FOR THREE OR FOUR MONTHS AFTER AN ATTACK OF SEVERE TYPHOID FEVER.

With regard to the symptoms generally of typhoid fever, it is of great practical importance to be constantly alive to the fact that no necessary connection exists between the intensity of the general symptoms of disease and the extent of the intestinal mischief which may supervene, or the absolute danger of the case. Two cases, out of several related by Dr. Bristowe, show that the patients (men) carried on their daily avocations, so mild *seemed* the disease to be, up to the very moment of fatal perforation of the gut. Indeed, the most suddenly fatal cases seem to be the very cases in which strongly marked febrile phenomena do not occur. In a case related by Dr. Murchison, a man twenty-one years of age died on the twenty-fifth day of the fever. Up till the twenty-third day there were no symptoms to indicate danger. He suffered from very slight diarrhœa; the pulse seldom rose above 90; and the patient could get out of and into bed. About forty-two hours before death the pulse rose to 120, associated with sudden pain in the lower and right side of the abdomen. Profound collapse indicated that perforation had ensued, and death soon followed. The *very slightness of symptoms* ought, therefore, to rouse suspicion, knowing, as we now do, that, associated with the characteristic eruption, the following *four* sets of phenomena may be all that precede a fatal hemorrhage or peritonitis; namely,—(1.) An elevation of temperature towards evening of only 1° or 2° above 98° Fahr.; (2.) Moderate increase towards evening in the fulness and quickness of the pulse; (3.) A little headache during the first six days; (4.) Scanty urine.

Again, the physician must keep in view the fact that relapses of

all the symptoms, including the eruption, not unfrequently supervene. He must not be betrayed into the belief that danger is past, if, towards the eighth or tenth day, the little headache that prevailed may pass away, and the other febrile phenomena just mentioned may subside. It is on record that events such as these have led to the belief that convalescence from a mere "febricula" had been established, leading to the discharge of the unfortunate patient from hospital. His vocation, if a soldier, would then compel him to undertake severe duties during the actual height of a severe disease, made more dangerous and perhaps fatal by such a mistake.

Another symptom, often very painful, is *meteorism*, or the accumulation of air in the large intestine. This is present in a greater or less degree in one-half of the cases, and when considerable it always marks a grave affection, and one generally fatal. On the contrary, the abdominal muscles are, in a few cases, tense and strongly contracted. It is, however, the experience of all physicians that there is no condition so low, and no symptoms so severe, from which the patient may not recover; and, on the other hand, there is no case of this form of fever so slight that it is to be considered free from danger. The prognosis must therefore be cautious, because perforation of the intestine may follow the mildest case, and death from peritonitis ensue.

The symptoms of typhoid fever cannot be said to be fully expressed till the characteristic eruption has appeared.

The Eruption consists of the so-called *rose spots* peculiar to *typhoid fever*, the "*taches rosées lenticulaires*" of Louis. They begin to appear from the sixth or seventh to the twelfth or fourteenth day of the disease, very rarely later, and still more rarely at an earlier period than the sixth day. A very delicate scarlet tint of the whole skin, closely resembling the skin of a person soon after leaving a hot bath, sometimes precedes, by a day or two, the characteristic eruption of typhoid fever; and this is important to remember, because it may be mistaken for the rash of scarlet fever, especially if sore throat is present. The eruption consists of slightly elevated papulæ or pimples; but, to detect their elevation, the finger must be passed very delicately over the surface of the skin, because, although pimples, they are not hard, like the first day's eruption of small-pox. Their apices are neither acuminate nor flat, but invariably lens-shaped or rounded, and the bases gradually pass into the level of the surrounding cuticle. No trace of vesication can be detected on their apices. They are circular, and of a bright rose color, the color fading insensibly into the natural hue of the skin around. Their margin is never well defined. They disappear completely on pressure, resuming their characteristic appearances as soon as the pressure is removed. These characters they preserve from their first appearance to their last trace. They leave behind no pit, scar, or stain. They vary in size, but their usual diameter is nearly 2 lines, but varying from 1 to 2½. The ordinary duration of each papula is about two days, but its existence varies from two to six days, and fresh ones generally make their appearance every day or two after the first appearance of eruption, and they continue

to appear in successive crops till the twenty-first or twenty-eighth day of the disease.* Sometimes only one or two are present at first, after which one or more fresh ones make their appearance. The eruption of such spots does not consist of a great number at one time—only from six to twenty. The eruption occupies usually the abdomen, thorax, and back; but is sometimes present on the extremities, and is sometimes, though rarely, so thickly seated that scarcely an interval of normal cuticle is left between. THIS SUCCESSIVE DAILY ERUPTION OF A FEW SMALL, VERY SLIGHTLY ELEVATED, ROSE-COLORED SPOTS, DISAPPEARING ON PRESSURE, EACH SPOT CONTINUING VISIBLE FOR THREE OR FOUR DAYS ONLY, IS PECULIAR TO AND ABSOLUTELY DIAGNOSTIC OF TYPHOID FEVER.†

The eruption is, however, often so scanty that the physician may justly hesitate for a day or two to make a diagnosis. The first crop of the eruption is rarely quite decisive; but as soon as successive crops, even of two or three spots each, appear, all doubt is removed. When the eruption is scanty, it is advisable to surround each individual spot with an ink line, in such a way as to distinguish accurately the period of its appearance (W. T. GAIRDNER). It is the occurrence of this eruption which clinches the diagnosis; and which becomes absolute, as regards typhoid fever, when in a febrile disease, attended by diarrhoea, or simply looseness, unequivocal rose spots appear on the sixth or eighth day. If they do not appear, then the diagnosis cannot be said to be complete till the case has been watched for several days, and the age of the patient and the history of the illness has been fully and carefully studied. In children between *one* and *five* years of age the phenomena do not seem to be so easily observed as in adults.

[A miliary eruption frequently appears in typhoid fever, from the eleventh to the twentieth day, consisting of groups of small, transparent, prominent vesicles, like congealed tears (*sudamina*); its site is the front of the neck, epigastrium, chest, bend of thighs, and anterior part of arm-pits; but it may extend over the trunk and extremities. It must be looked for sidewise and very near. Its diagnostic value is slight, as it is to be found quite as often in acute rheumatism, pyemia, puerperal fever, &c.]

Occasionally we meet with clear, pale-blue, or slaty-colored oval spots (PIEDAGNEL, FORGET, DAVOST), like partially effaced ink-stains (*taches ombrées*, *taches d'encre*, *taches bleuâtres*), not prominent, sometimes even apparently slightly depressed, unaccompanied with itching, and not af-

[* Though the abundance and persistence of the eruption have no relation to the severity of the attack, yet when it reappears in successive crops, an aggravation of the general symptoms will be noticed with each recurrence.]

[† Though far from contesting the semeiological value of the papillary rose eruption in typhoid fever, there is no doubt that it is often wanting. In 70 cases Chomel found it absent in 16, though carefully looking for it in every stage. In 70 cases observed by Flint, the eruption existed in 49, and he found the proportion varied in different years. In some epidemics they are common, in others rare, or wanting. Trousseau states that they have never been observed in any epidemic of typhoid fever in Touraine (France). Racle, *Traité de Diagnostic Médical*, Paris, 1864, believes antiphlogistic treatment at the outset of the disorder prevents their development, and states that, for this reason, they are rarely met with in the wards of Dr. Bouillaud in La Charité at Paris.—EDITOR.]

fectured by pressure. They form and disappear slowly, last for some time, fading one day and deepening the next; few in number, from four to ten, they are found on the abdomen, upper part of thighs, base of thorax, and rarely on the extremities. The time of their appearance is variable, being sometimes as early as the first week. They would seem to be more common in certain epidemics than in others. Some observers have regarded them as a species of ecchymosis, and the first stage of petechiæ, and connected with the blood lesion; but one of many objections to this view is that when present it is constantly in light cases. Though, it is believed, peculiar to typhoid fever, their rarity takes from their diagnostic importance.]

Typhoid Fever in Children.—It has been now clearly established that typhoid fever is by no means an unfrequent disease amongst children, and has been often described under the name of "*Infantile remittent fever*." [It occurs sometimes epidemically. This has been noticed at the Children's Hospital at Paris, and Dr. Rilliet saw an epidemic typhoid fever in a small village near Geneva (Switzerland), which attacked children only.] Boys seem to be more liable to attack than girls. [Of 121 cases recorded by Rilliet and Barthez, 81 were boys and 31 were girls; and of 121 observed by Taupin, 86 were boys and 31 girls.] It is most frequent between six and eleven years of age; and from five to nine seems the period of greatest liability. Its occurrence is rare during the first years of life. Nevertheless, it is on record at the following very early ages: namely, between two and three months; three months; six months; seven, ten, and thirteen months (WUNDERLICH, HENNIG, FRIEDRICH, RILLIET). The author of a very interesting Review on the typhoid fever of children, in the *British and Foreign Medico-Chirurgical Review* for July 1858, p. 161, mentions, in his own experience, the occurrence of typhoid fever in a girl one year and seven months old; and also in a boy two years of age.

The chief symptoms of typhoid fever in the child are,—[epistaxis.] splenic enlargement, diarrhœa, meteorism, gurgling in the course of the colon; associated with pyrexia, quickened respiration, bronchial catarrh; delirium, somnolency. [The tongue, though dry, is rarely fissured or cracked; retention of urine is infrequent; vomiting, particularly at the outset, is common. Peritoneal perforation, intestinal hemorrhage, and gangrene of the intestine, seldom happen; otitis often occurs.] The eruption already described, and sudamina, are nearly constant in children after five years of age. The rose-colored spots are especially frequent on the back and the extremities, so that, if the abdomen and chest only are examined, their presence may often not be apparent.

The Temperature during Typhoid or Intestinal Fever.—Wunderlich has given a summary of results derived from the observation of 700 cases of typhoid fever, investigated thermometrically (*Arch. der Heilk.*, vol. ii, 1861, p. 433; also, *Edin. Med. Journal*, Nov., 1862, p. 465).

The course of the disease is typical, and the type is characteristic; and when irregular cases occur, irregularity may in general be traced to a special cause. The mode of accession is pretty nearly the

same as in much more severe cases. Increase of temperature in the evening and remissions in the morning follow one another for about three days, the temperature every morning and every evening being about 2.2° Fahr. higher than on the preceding morning and evening, while the morning temperature is generally about 1.1° lower than that of the previous evening; or, according to the following formula:

First day, morning, 98.5° ; evening, 100.5° ; *second day*, morning, 99.5° ; evening, 101.5° ; *third day*, morning, 100.5° ; evening, 102.5° ; *fourth day*, morning, 101.5° ; evening, 104° . In the second half of the week the evening temperature is from 103° to 104° ,—the morning temperature about a degree lower. On the third or fourth day the fastigium or height of the fever is attained, when the temperature in the evening amounts at least to 103.5° Fahr. From that time onwards the fever proceeds in regular stages of weekly and half-weekly periods. When the temperature on the first or second day reaches to 104° , or where in a child or in an adult the evening temperature between the fourth and sixth days does not rise to 103° , where, in the second half of the first week there is considerable abatement of the evening temperature, we have in such cases certainly *not to do with typhoid fever*; on the other hand, the disease may always be recognized when there is in the evening hours a persistent elevation of temperature. During the second half of the first week both mild and severe cases follow the same course, so that, for the purpose of prognosis, the determination of temperature is of little consequence during the first week. In the second week typhoid fever may be excluded with the greatest probability, if between the eighth and eleventh days the temperature is below 103° . Such a temperature is rarely met with at this period in any other disease, and where it occurs unequivocal symptoms will certainly be present. It is only in the maxima of the temperature that sometimes a difference is visible between very mild and very severe attacks. In the mild cases there is now and then a large decrease of temperature observable towards the end of the first week—namely, from 105.8° to perhaps 102.5° . *At the beginning of the second week*, or at the latest during its second half, severe and mild cases diverge so unmistakably that the course at that period is decisive as regards what the future progress will be. A favorable course during the second week permits us to anticipate a favorable termination of the disease. In mild cases (analogous to those of modified small-pox), although the evening temperature may reach 103° ; and even exceed 104° , considerable abatement (1° to 2°) takes place during the morning, which becomes more and more obvious towards the end of the second week. Such mild cases progress favorably when the exacerbations do not begin before ten o'clock in the morning, so that before midnight an abatement takes place; when these conditions remain daily the same, or when a diminution of temperature shows itself, although not more than half a degree; and, lastly, when there is an abatement on the eleventh, twelfth, and fourteenth days.

when a fall in temperature about the middle of the week does not take place.

A permanent temperature of 104° is an unfavorable sign—so also is an elevation of the morning above the evening temperatures. A severe form of the disease is to be expected when the morning temperature at the beginning of the second week is above 104° , and when the evening reaches nearly 106° ; and when towards the end of the week a rise still takes place. The most unfavorable cases are those where, in addition to these unfavorable conditions, oscillations are added, even if these consist in diminution of temperature.

In the third week the patient enters upon those highly characteristic quotidian vacillations of 4° , 6° , and even more degrees Fahr. between the morning and the evening temperatures. If the case is mild, the evening exacerbations gradually decrease in intensity, and the morning temperature is regularly at first from 3° to 4° below the evening. The fever ceases in the course of the week, the temperature reaching its natural standard, and convalescence commences, as a rule, sometimes in the third week, generally in the fourth week, or at the latest in the fifth week.

In severe cases the characteristics mentioned as peculiar to the third week already commence in the second. The temperature in the morning is high (104° Fahr., and more), and differs but little from that in the evening; or even that high temperature increases in the afternoon and evening to a still higher degree. In this it differs from a remission of the fever in a mild case, inasmuch as in remissions the heat in the mornings sinks below the average degree of the temperature in typhoid cases—*i.e.*, below 103.3° Fahr. to 104° Fahr. In severe cases, on the contrary, the temperature always remains above the average degree, and rises still higher in the evening. Real remissions in such cases are not met with during the whole of the second and third weeks; but when the case is favorable, although severe, the temperature is about a degree lower than in the second week, and the remissions do not take place till the fourth week; and if the temperature remains as high, or rises higher than it was in the second week, the remissions do not occur till the fifth week, and irregularities in the ranges of temperature always render the prognosis doubtful.

So late as *the fourth week* the evening temperatures are still high, and they decrease very gradually even in favorable cases. Towards the end of the fourth week, or in the fifth week, or even so late as the sixth week, the great and increasing remissions commence—a period at which various other phenomena occur, and when the complications and dangers are numerous.

The complications generally make their appearance about the third week, and threaten to tend to a fatal end up to the very beginning of convalescence. In the mild types the growths in the intestinal glands are no doubt such as are eliminated by mere rupture of the vesicles, which simply heal without ulceration. The severe cases owe their severity partly to the more extensive growth of new material in the vesicles of Peyer's glands, partly to the mode of elimination of that material; the healing of the parts being ac-

complished under great excitement of vascular reaction, renewed hyperæmia, sloughing, softening, and final cicatrization.

Cases intermediate in severity between the mild and severe cases just described are not unfrequently met with. Many of them, although they show a course more or less irregular, nevertheless follow a pretty clearly defined type as to variations of temperature, and are capable of clinical recognition. There are, still considerable evening exacerbations during the second week, yet with a tendency to abatements in the mornings. During the third week great vacillations between morning and evening temperature continue, and sometimes also between single days. During the fourth or fifth week the normal temperature is reached in the morning; but it is only in the fifth or sixth week that the temperature becomes permanently normal—the evening temperature showing a complete freedom from fever—so that the beginning of convalescence can only be established with certainty by the use of the thermometer.

In the majority of cases of typhoid fever, severe as well as mild, a peculiar periodicity of weeks and half weeks cannot be mistaken. Each week shows a distinct character, which cannot be overlooked in a graphic representation. On the first and last days of each week changes generally take place which are either temporary changes or continue till the fever subsides.

Duration of Attack and the Mode of Recovery, or the transition into the feverless state, is peculiar and characteristic of enteric fever. With rare exceptions, the defervescence is a remittent one. The great vacillations between morning and evening recur for a longer or shorter interval. For weeks the evening temperature may amount to 104° Fahr. or more, whilst in the morning the patient is quite free from fever. At the same time the transition into the feverless condition may follow different courses. The remissions may either become longer and longer—the morning temperature decreasing and the evening remaining stationary; or after some time the remission may become shorter and shorter—the evening temperature, together with the morning temperature, gradually descending. Again, the differences between the morning and the evening temperatures may remain nearly the same, while a relative decrease takes place at both periods; or the fever shows a sudden transition into the remissions with low temperatures—changes which generally correspond with the commencement of weeks. The period of development of the disease occupies two weeks, or a week and a half in slight cases; in severe cases it may occupy two and a half to three weeks. The initial stage (that is, the period when the growth of material in Peyer's patches takes place) lasts about half a week. The removal or elimination of the growth may take place in a week; but the process may extend over several weeks. In mild cases the disease continues at its height for only a week or a week and a half,—rarely for two weeks; so that the whole duration of a mild case of typhoid fever extends from eleven to eighteen days. The period of convalescence occupies from one to two weeks. The whole disease, therefore, in mild cases may be

gone through in from three to four weeks,—rarely in two weeks and a half.

In severe cases the disease continues at its height for from two weeks and a half to three weeks and a half. Then an undecided period of irregular duration succeeds, after which decided abatement is established, the defervescence occupying a week, followed by another week of convalescence. Consequently, the whole disease extends from four and a half to ten weeks, or even longer. Regarding the mean duration of illness in *typhoid fever* considerable differences of statement are to be found—a circumstance not to be wondered at when the nature and seat of the pathognomonic lesions of this form of fever are recognized as influencing the duration of the disease.

Dr. Shattuck assigns the mean duration of <i>typhoid fever</i> to be	22 to 24 days.
The mean duration of the Parisian cases of 1839-40 were	19.6 "
Dr. Jackson's experience in America gives	22 "
Dr. Jenner's experience in London leads him to give	21 to 30 "
Dr. Murchison	24.6 "

The mean of these varied statements gives nearly twenty-three days.

It is now well known that during the progress of this form of fever there is a repetition of the development of new material in the individual gland-vesicles of the intestine, and consequently a succession of retrograde metamorphoses; so that, in many extreme cases of *typhoid fever*, it is not unusual to have the malady prolonged throughout a course nearly double as long as that of *typhus*; and that, undoubtedly, the influence of the secondary local lesions of *typhoid fever* is great in protracting the disease. Thus it is that a very indefinite idea of its duration prevails; and, as Dr. Jenner has shown, it is of the greatest importance to know when the original fever ceases, after which we are to consider the subsequent symptoms as due to the effects produced by the local lesions.

As long as fresh eruption continues to appear, the fever cannot be regarded as having terminated; and, except in cases of relapse, fresh spots never appear after the thirtieth day (JENNER) or thirty-fifth day (MURCHISON).

True relapses are occasionally observed. They occur about ten days or a fortnight after convalescence from the first attack, and are marked by a return of all the former symptoms; while the duration of the attack is usually shorter than that of the first; and, according to the experience of Murchison, it is more severe. Such relapses are most common in autumn.

In particular cases following a spontaneous course, and still more in cases treated with calomel, a considerable shortening of the whole febrile period will not only be observed, but some peculiar modes of defervescence will occur. The temperature is reduced where calomel acts beneficially; and the beneficial remission is persistent.

The influence of hemorrhage from the bowels in reducing temperature has been also well shown in a case recorded by Dr. Parkes.

It occurred in a female twenty-five years of age. Diarrhœa was considerable; and blood was largely passed in fluid stools the night before the seventeenth day of the fever. On the morning of the seventeenth day the temperature was as low as 93° Fahr., rising in the evening to about 101° Fahr. After the eighteenth day diarrhœa ceased; but the differences between the morning and evening temperatures continued to be very great; and it was not till the twenty-sixth day that these differences began to grow less and less.

The approach of death is indicated by a permanent or persistent elevation of temperature in the morning (as high as 106°); by a sudden rise to 108°, or even higher; and more seldom to a depression below 93°.

Condition of the Urine in Typhoid Fever.—It is not till the third or fourth day of the fever that the urine assumes any special characters. It is peculiar in the following respects:

I. As to normal constituents: (1.) *The water* is greatly diminished, generally about one-half or even to one-fourth or one-sixth. This lessening of the water is most marked during the first week; it then begins to increase gradually during the second and third weeks; and at the end of the fourth week, in favorable cases, it has reached its normal standard. (2.) *The whole amount* of the urine does not seem to stand in any close relation to the febrile heat; but when the temperature begins to fall permanently, the urine increases at once, or very soon after. (3.) *The specific gravity* of the urine is high in almost all cases where the urine is scanty; and at convalescence the specific gravity diminishes, sometimes before the amount of water increases; i. e., at convalescence the lessening of the solids of the urine is often prior to the increase in the water. (4.) *The urea*, as a rule, seems to be augmented, during the febrile period, above the physiological standard proper to the individual; and it sinks again below this standard during convalescence. The amount of increase varies: Vogel has noted 78 grammes, or 1200 grains, in 24 hours; while Parkes has noted 57 grammes, or 880 grains, in that time. In most of the cases observed by Dr. Parkes the average increase has been about one-fifth above the physiological standard proper to the individual; and the augmentation is most marked in the first week, when the water and the chloride of sodium are at the lowest point; and if the fever be continued beyond the third or fourth week, the urea keeps up in amount. The relation of urea to temperature is yet uncertain. (5.) *The chloride of sodium* is diminished (indefinitely); the cause of the diminution being in part due to the lessened ingress of this substance on account of spare diet: or due to the elimination of large quantities of it with the stools or the sweat. (6.) *The uric acid* is uniformly increased in amount; and it is relatively greater than that of the urea. It is often doubled in amount; and the increase progresses up to the fourteenth day, when it is at its greatest. It then diminishes to the twenty-first or twenty-eighth day; and during convalescence falls below the normal amount. Spontaneous deposits of urates occur very frequently; and when there is no such deposit it may be brought about by a drop of acid; but as yet the

fact has no particular significance. (7.) *The sulphuric acid and phosphoric acids* maintain their amounts very much the same as in health; and sometimes a little above that: and seeing that much of the former is derived from food, its abundance in typhoid fever would indicate active tissue-change, when little or no food is being taken. (8.) *The pigment* at first is sometimes enormously increased, measured after Vogel's method (by comparison with a scale of colors). It has sometimes amounted to 80 or 100 in 24 hours, the normal amount being 3 to 6 (VOGEL). This, Dr. Parkes says, is to be referred to increased disintegration of blood-cells: it is therefore much more highly colored than the mere concentration will account for. (9.) *The acidity of the urine* appears always great during the early period, simply from concentration; but by neutralization with an alkali it is found actually to be below the average by one-fifth, or even by one-fourth. During the third week the acidity still continues to lessen; and ultimately the urine may even become alkaline from fixed alkali. It may also become alkaline from ureal decomposition, soon after being passed. Therefore it is necessary, in all observations on this point, to distinguish carefully between the alkalinity due to fixed alkali and that due to ammonia.

II. As to abnormal constituents: (1.) Albumen occurred in 33.3 per cent. of the cases examined by Dr. Parkes. In 23 per cent. of these cases it was temporary, and entirely disappeared before the patients left the hospital. In the other cases it was permanent; and in one of these a very profound kidney lesion which had not previously existed was immediately excited by the fever. (2.) Renal epithelium, casts, and blood are sometimes seen in the cases with temporary albuminuria—the blood generally in microscopic quantities; although in bad cases it may be greater in amount. Different phenomena in the course of typhoid fever variously affect the urine. The effect of diarrhœa is to diminish both water and solids, the chloride of sodium especially. Non-excretion of urea, or deficiency in its solid matters, often coincides with the putrid, adynamic, or profound "typhoid" state, and with symptoms which imply more or less blood poisoning from retention. Local lesion in the kidney may lead to this, or, from failing circulation, less blood may pass through the renal vessels, or there may be, as Dr. Parkes suggests, some special condition or combination of urea which hinders transudation. Such non-excretion is most apt to supervene during the third or fourth week, when the first stage of the disease is over, and when the growths in Peyer's glands, and in the mesenteric glands, are softening, when the secondary blood poisoning occurs, and when the heart's action tends most to fail.

Judging from the urine alone, the febrile action appears strongest in the first week of enteric or intestinal fever (typhoid), although the temperature is highest in the second and commencement of the third week.

The prognosis in severe typhoid fever appears to be more favorable in proportion to the free excretion of urea and uric acid (PARKES). The excretion of these effete products is a most necessary point; for there is more danger in the retention than in any

amount of fever and *formation of them with elimination*. The greater the excretion in typhoid fever the better; and as long as 500 to 700 grains of urea in men, or 300 to 500 in women, are being passed in each twenty-four hours, the progress so far is favorable. But whenever, while the fever continues, the urea falls much below these amounts, we may anticipate a low typhoid condition, or some local inflammation, as pleurisy, which may relieve the blood for a time from some of the effete products, but which at the same time may kill the patient.

The existence of slight albuminuria or hæmaturia is not of itself unfavorable; but if either be in large amount, or if there be exfoliation of epithelium or renal cylinders present in the urine, retention of urea and its consequences may be expected.

Morbid Anatomy of the Lesions in Typhoid Fever, with special reference to the Phenomena and Progress of the Disease.—The abdominal complications of typhoid fever, as they are sometimes called, are mainly due to lesions of the solitary and aggregate glands of Peyer, and to enlargement of the mesenteric lymphatic glands. This lesion in the ileum is especially recognized as the “anatomical sign” of enteric or typhoid fever. It is necessary to remember, however, in connection with the age of typhoid fever patients, that the solitary vesicles and the aggregate glands of Peyer are known to be most fully developed and most active in youth, up to the age of early manhood; after that time they begin to disappear, and are obviously less active in the adult after thirty years of age. Structure and function seem to be alike impaired by age, till at length, after forty or forty-five years, traces only of their existence are apparent, or they have altogether disappeared. The gland substance (whose structure has been so well described by Dr. Allen Thomson, Kölliker, and Boehm) no longer exists; and the places where the patches of Peyer once were may be detected only after careful examination,—a mark of varied form and character being all that indicates the place of the patch. There is therefore a good anatomical reason why typhoid lesions are rarely found after fifty years of age, and seldom after forty. Dr. Jenner records only three cases beyond fifty—namely, one at fifty-one and two at fifty-five. Dr. Wood has observed one case at fifty-five years of age. Dr. Murchison notes two cases above sixty-five, and refers to five other cases between sixty and seventy-five, related by MM. Lombard and Gendron. Dr. Wilks refers to the case of a woman aged seventy, of very doubtful history (*Path. Society*, 1861). These exceptional cases are explicable when it is known that the existence and functional activity of these glands are sometimes prolonged for an indefinite term of years beyond the usual period of their existence. On the other hand, it is in childhood and early life that these glands are most obvious, and their functional activity the greatest; and therefore it is extremely significant to find that “more than one-half of the cases of typhoid fever occur between fifteen and twenty-five years of age; and in very early life the proportion of cases of typhoid would be greater were it not that many children laboring under this disease are described as cases of ‘*Infantile Remittent Fever*’” (MURCHISON).

The following records with regard to the age of typhoid fever patients, collected by Dr. Murchison, demonstrate these points:

PERCENTAGE OF CASES OF TYPHOID FEVER AT DIFFERENT AGES.

Under ten years,	6.04
" fifteen years,	20.14
From fifteen to twenty-five years,	52.08
Twenty-five years and upwards,	27.76
Thirty,	14.22
Forty,	5.19
Fifty,	1.46
Sixty,	0.5

The average age of typhoid fever cases is $21\frac{1}{2}$; and the fever is pre-eminently a disease of childhood and adolescence.

Lesions in Typhoid Fever.—Of these the most noticeable are to be seen in the intestines, and may be considered in the following stages:

I. *A generally congested state of the mucous membrane of the intestines, especially expressed in the vicinity of the solitary glands, which are surrounded by vascular rings, and clustered groups of vesicular glands which constitute Peyer's patches.** This vascularity seems to be very general, involving more or less of the abdominal viscera. Sensations of heat and abdominal distress are associated with this morbid state, and the lineaments of the belly are obliterated.

II. *Associated with this congestion, the gland-vesicles become obviously prominent.* Increased growth of the gland-cells occurs, till the closed sacs of the glands become filled up with crude material. This condition is sometimes described as *intumescence* of the glands; and with the congestion just noticed constitutes the stage of "*infarction*," as described by the older authors. Symptoms denoting

* The following tabular statement of the anatomical forms of the glands which compose the substance of the mucous membrane of the alimentary canal is mainly condensed from a careful description of them by Dr. Allen Thomson, Professor of Anatomy in Glasgow, published in Goodsir's *Annals of Anatomy*, vol. i, p. 33, and from the descriptions of Kölliker. The nomenclature is definite, and distinctive of the various forms of the glands; and it will be adhered to in all the future descriptions of lesions of the mucous membrane of the intestines. Much confusion prevails from the indifferent use of the terms vesicle, tubule, follicle, &c., as applied to the mucous glands in descriptions of the lesions in dysentery and fevers; therefore it is considered necessary to explain exactly, at the outset, the nomenclature adopted in the text.

I. VESICULAR, LENTICULAR, OR PIMPLE-LIKE GLANDS—Usually closed.

(a.) Solitary—*e. g.*, in the palate, buccal membrane, œsophagus, and stomach; also found deeply imbedded in the great gut, and scattered more near the surface of the small gut.

We know nothing about the comparative abundance of these glands in a healthy intestine; nor are we certain whether or not they disappear after a certain age, like the vesicles which compose Peyer's patches. Many of the so-called solitary glands seen in disease may be in reality new formations. At all events, they occur in much greater numbers in certain diseases than their known frequency of appearance in the healthy intestine would lead us to expect.

(b.) Clustered in groups—*e. g.*, Peyer's patches of glands in the ileum.

II. FOLLICULAR OPEN GLANDS OR CRYPTS—A transient condition of the vesicular glands, after rupture and discharge of their contents—*e. g.*, great gut and stomach.

III. TUBULAR GLANDS—Occur in the small and large intestines, as the so-called follicles of Lieberkühn; and in the stomach, as the stomach tubes.

IV. RACEMOSE GLANDS—Consisting of tubes with simple sacs or vesicles, in clusters round a stalk or duct—*e. g.*, the cardiac œsophageal glands, and the duodenal glands of Brunner.

intense irritation of the mucous membrane—*catarrhal* and *gastric* symptoms—prevail; and the mucous membrane generally is swollen and turgid, especially the villi of the intestines, which are particularly distinct, imbedded in a thick layer of dirty-yellow gelatinous mucus. Although these conditions seem to involve the whole of the mucous membrane in the first instance, yet they soon begin to be more expressed towards the lower end of the small gut than in any other part. The time at which this increase of cell-growth commences in these glands is not yet well defined in relation to the day of the fever. It seems certain, however, that it occurs within the first week; but it may be later. A case is described by Dr. Sankey, in the first volume of the *Pathological Society's Transactions*, in which dissection showed the growth of the glands as early as the fifth day. The bulging of the patch and the extent of intumescence vary considerably in different patches; and simultaneously with these conditions the mesenteric glands begin to increase in size. They, too, are supplied with an increased quantity of blood, and the increased tissue of the gland becomes unusually soft and elastic.

III. *A subsidence of the general congestion, and of the generally turgid state of the mucous membrane, takes place after the gland-growth has been fully developed.* Nevertheless, the growth continues actively, and progresses rapidly till the patches of Peyer become so thick as to be elevated three or four lines above the surface of the mucous membrane. A beautiful vascular halo encircles them, stopping short at their margins; and a contracted border surrounds the margin of the patch, which gives it a sessile fungiform aspect, with an umbilicated-like depression on its surface. Growth is now confined within narrow limits, pressing on the muscular coat below and the mucous coat above. The patches assume various aspects as to color; and, when vascular, they have an appearance which has acquired for them the description of being like "fleshy lumps;" their tawny gray color showing through the peritoneum of the gut. Varicose vessels abound in the vicinity,—a fact of some importance in connection with the formation of thrombi, and which may lead to hepatic or pulmonary embolism. The specific gravity of the mucous tissue of Peyer's patches is obviously changed by such increased growth, ranging from 1.032 to 1.044.

IV. *Softening of the contents of the tumid gland-cells* seems to be the next event in the series, and which would appear to be preliminary to one or other of the following results, namely:

V. *Conditions under which the softened contents of the glands begin to be eliminated.* This elimination seems to take place in one or other, or in each of the three following ways; conveying out of the body by the intestinal discharges abundance of morbid material, presumed, with great probability, to contain the specific virus of the fever:

1. *Elimination without ulceration—simply by the rupture of the hitherto closed gland-vesicles.* This is the usual and natural way in which the vesicles of Peyer's patches become open follicles in the course of their normal physiological existence. For many reasons I am induced to believe that this is the natural, the most common,

and the most frequent mode by which the softened new growth in typhoid fever is got rid of—namely, by the escape of the softened contents of the glands through the rupture of the vesicles in the ordinary way. The vesicles then collapse, and assume the appearance of little pits, depressions, or follicles, and so give rise to that “reticulated indistinctly pitted surface” so often seen after all evidence of gland-structure has disappeared. This view is also consistent with the observation of Wedl, when he says that “the glands in question not unfrequently burst; and the capsules also may collapse, in consequence simply of absorption of their contents. Owing to one or other of these occurrences, the Peyerian patches acquire the well-known reticulated aspect, since the mucous membrane surrounding the individual capsule assumes the form of a projecting border; and entire patches present the appearance of a fine sieve” (*Pathological Histology*, p. 221). The ordinary peristaltic action of the intestines may assist this mode of elimination by rupture of the vesicles, if the softening is complete. One case I dissected at Scutari led me first to this conclusion. In this case a process of growth and elimination seemed to have gone on for at least one month previous to death; and the elimination of the material from the patches took place without ulceration. The man died suddenly from aneurism of the aorta. A lull in the febrile symptoms had led to his premature discharge from hospital; and the day on which he suddenly died was to have witnessed his embarkation for England. Peyer's patches were in an extremely interesting condition. They were all large and obvious. In some parts of them the vesicles were greatly distended with the material of growth, in a milky-like condition; while other parts of the same patch were completely bare, and dotted over with minute points of black pigment. These parts were quite bare of all gland-structure, and had a reticulated appearance.

In corroboration of this view, it is to be observed, further, that Dr. Friedrich, of Dresden, considers the elimination of the deposit from the Peyerian patches in the typhoid fever of children by the formation of sloughs and ulceration, as extremely rare. For the most part, only single follicles [vesicles?] in the glandular assemblage are infiltrated, and these, either from resorption of the infiltrated material, or more often from rupture of the follicle [vesicle] within the intestinal canal, revert to a normal condition without the formation of any cicatrix (*Brit. and For. Med.-Chir. Review*, July, 1858, p. 162.)

2. *Elimination by ulceration of the swollen gland-vesicles* in groups of various sizes, involving more or less of surrounding tissue in ulceration, and tending to induce perforation of the gut or peritonitis. Considering the severe nature of this lesion, and taking into account the fact that a large proportion of cases of typhoid fever recover, it seems to me that this is a mode of elimination which occurs much less frequently than the mode already described. Of course, it is the state most frequently met with after death; for ulceration of Peyer's patches is the characteristic “anatomical sign” of typhoid fever. The time of commencing ulceration of the

mass appears to be about the ninth or tenth day (MURCHISON); and the softening which precedes ulceration is associated with a return of the violent congestion to the small intestines, when the veins especially are filled with dark-colored viscid blood. The outbreak of the ulcers is always characterized by an aggravation of the original symptoms, after it may have been sanguinely supposed that convalescence had decidedly taken place. But in such deceptive convalescence the abnormal temperature is maintained, showing with absolute certainty that the fever is not at an end. The ulceration therefore is usually denoted (*a*) by a re-accession of the febrile phenomena, with or without diarrhœa; (*b*) by abdominal pains and tenderness. Judging from post-mortem examinations, the ulceration seems to commence at the lowermost patches in the glands nearest to the cœcum, and the ileo-cœcal valve is often implicated in the destruction. The ulcers vary in number and in extent; and although there is a tendency to perforation of the gut in fatal cases, yet actual perforation is not common, and peritonitis may supervene without perforation having actually taken place. Various statements have been made concerning the tendency of typhoid ulcers to perforate the gut, and the frequent association of this lesion with peritonitis. Perforation is said to be rare in the northern parts of Europe (HUSS); but, from the records of Drs. Murchison and Bristowe, it appears to be a more frequent mode of fatal termination than has been commonly supposed in this country. Of fifty-five fatal cases, perforation occurred in eight (LOUIS); of fifteen fatal cases, perforation occurred in three (MURCHISON); of sixty-three fatal cases, perforation occurred in twelve (London Fever Hospital Records); of fifty-two fatal cases, perforation occurred in fifteen (BRISTOWE).

From these data it appears that perforation occurs in about one in five fatal cases; and it generally takes place through the ileum near the valve. Post-mortem examination often discloses vigorous attempts on the part of neighboring structures to limit by union and adhesion the results of perforation, obviously indicating, in practice, the necessity of absolute rest throughout the disease.

The characters which distinguish the ulcers of typhoid fever from other ulcers of the intestines may be stated as follows: (1.) They have their seat in the lower third of the small intestine, their number and size increasing towards the ileo-cœcal valve. (2.) They vary in diameter from a line to an inch and a half; but a number of ulcers may unite to form a mass of ulceration several inches in extent. Such extensive masses of ulceration occur close to the cœcum. (3.) Their form is elliptical, circular, or irregular—elliptical when they correspond to an entire Peyer's patch, circular when they correspond to a solitary gland, and irregular when they correspond to a portion of a Peyer's patch, or when several ulcers unite to form one. (4.) Elliptical ulcers are always opposite to the attachment of the mesentery. (5.) The ulcers never form a zone encircling the gut, as may sometimes be seen in the case of the tuberculous ulcer, but their long diameter corresponds to its longitudinal axis. (6.) Their margin is formed by a well-defined fringe of mucous membrane, detached from

the submucous tissue, a line or more in width, and of a purple or slate-gray color—an appearance best seen when the bowel is floated in water. (7.) After separation of the slough there is no thickening or induration of the edge of the ulcer, as in the case of the tuberculous ulcer. (8.) Their base is formed by a delicate layer of submucous tissue, or by the muscular coat, or occasionally by nothing more than peritoneum. (9.) There is no deposit of morbid tissue or new growth at the base of the ulcer; although sometimes fragments of yellow sloughs may be seen adhering to both the base and edges (MURCHISON, p. 547). The ulcers also are known to heal. Their cicatrices have been seen four, five, and thirty years after known attacks of typhoid fever (ROKITANSKY, BARRALLIER). And in cases where death occurs during a relapse, the cicatrices from the first attack may be found coexisting with the fresh growth in the vesicles, and with the recent ulcers of the relapse. As a rule, the reparative process does not commence till the end of the third week of the disease, and in one case, where the primary fever lasted three weeks, and where death occurred from complications about the fortieth day, all the ulcers in the ileum were cicatrized (MURCHISON).

Cicatrization commences by the growth of a thin, delicate, shining layer of new growth which covers the base of the ulcer, and is also attached to the basement membrane of the mucous coat. The fringe of mucous membrane becomes adherent to this new tissue, from the circumference towards the centre, until the healthy mucous membrane merges insensibly into the serous-looking lamina. The new film of membrane cannot at first be moved upon the subjacent coat, but after a time it becomes movable, and according to Rokitansky, even becomes covered with villi (MURCHISON). There is no evidence of the vesicular gland-structure ever being restored. The resulting cicatrix is slightly depressed, firmer, less vascular, and smoother than the surrounding mucous membrane. The bowel appears thinner at this part when examined by transmitted light. The depressed spot is never surrounded by any puckering, nor does it ever cause any diminution in the calibre of the gut.

3. *Elimination of the typhoid growth by sphacelus* of large masses of Peyer's patches. The whole gland-substance implicated is involved in the destruction. The cell-growth in the vesicles suddenly becomes so excessive that a condition is at last reached which is incompatible with the maintenance of life. The growth actually chokes itself; and the whole mass, or a great part of it, softens and dies. Such sphacelus has been known to happen as early as the twelfth day; but the process is generally more slow. A dirty yellow-brown slough forms, varying in thickness, and sometimes extending as deep into the substance of the gut as to expose its muscular layer on separation of the mass. There is a tendency to bleeding on separation of the sloughs; and such hemorrhage occurs in about one-third of the fatal cases. The frequent repetition of such hemorrhages during life has a marked influence in modifying the febrile phenomena. For example, in a case described by Dr. Parkes, in which the temperature was very carefully recorded three times daily, it was observed to fall *below* the standard of health on the fourteenth and fifteenth

days slightly, on the sixteenth day to the extent of 4° below 98° , on the seventeenth day to the extent of 5° , on the twentieth day to the extent of 2° . These falls of temperature were all traceable to the influence of repeated hemorrhages from the bowels. The occurrence of hemorrhage is always a most alarming symptom, and is most frequent during the third and fourth weeks of the disease. It varies in amount from a mere stain to a large quantity of blood, sometimes discharged in clots, and generally of a red color, in consequence not only of the rapidity with which it is passed out, but also, as Dr. Parkes has shown, in consequence of the alkaline reaction of the contents of the intestine. It may cause immediate death by syncope; or, by reducing the temperature and strength of the patient, he may sink exhausted, unable to cope with the disease. Whenever, therefore, blood appears in a case of typhoid fever, it is certain that the lesions of Peyer's patches are severe.

In addition to these three modes of elimination of the new growth from the intestinal glands, there are reasons for believing that it may be occasionally *reabsorbed*; unless such cases where resolution, independently of ulceration, commencing about the tenth day, may not be explained by the first method of elimination I have described.

VI. The mucous membrane of the intestines having existed for several weeks in a state of irritation which has been described, and the catarrh being more or less excessive, *an atrophic condition of the intestine at last supervenes*. The mucous tubes become wasted, irregular in form and size, sometimes separated by an interstitial growth of a granular nature, their bulbous ends disappear, and the whole substance of the gut becomes so thin that it resembles a portion of thin paper rather than intestine.

The mesenteric glands are invariably enlarged. They begin to enlarge at the very commencement of the disease, and sometimes attain a very large size, and their stages of congestion, of swelling, and of subsidence, go on simultaneously with the similar changes in Peyer's patches.

The spleen is usually greatly enlarged, varying from five or six to fourteen ounces, with a specific gravity varying from 1052 to 1059. Its Malpighian sacculi (glandular) are also intumescent.

Pulmonary lesions occur (1) as infiltrations, or (2) as the consolidation of pneumonia, or (3) as portions of lung which have become carnified.

In the first-mentioned form of lesion the growth seems to commence in the terminal air-vesicles, ultimately assuming the form of a miliary deposit, with a semi-transparent gelatinous appearance. It is the irritation set up by this sudden growth which generally gives rise to pneumonic consolidation. Softening and friability of the pulmonary texture is thus a very constant post-mortem state in protracted cases of typhoid fever. Such lesions usually supervene during the later period of the fever, and when the ulcerations of the intestines are extremely spread (Huss). In this respect only it differs from the consolidation of the lung to be described in typhus fever. This lesion has been also termed *non-granular consolidation*, dependent for its origin and development on a specific cause; and

it may be observed not only in the course of *typhoid fever*, but in *measles*, *scarlet fever*, and *small-pox*. A portion of the lung in this condition has a mottled aspect. There are patches in it here and there, varying in size from a single lobule to half, or more than half, of a lobe, of a deep bluish-chocolate, violet, or purplish-slate color, bounded by a well-defined angular margin, and crossed and mapped out into smaller patches by dull, opaque, whitish lines. These are seen to be thickened into lobular septa. Scattered in the midst of the larger patches, one or more comparatively healthy lobules are frequently found, of a pale brightish-pink color, contrasting strongly with the hue of the surrounding tissue. The pleura which covers the part may have a slight milky-like aspect (DR. JENNER). It is also extremely probable that much of these thoracic lesions in such cases may be due to the direct passage of fibrinous particles from the large veins surrounding the diseased intestinal glands (in the case of typhoid fever); for clots thus tend to form in the bloodvessels, near the site of irritation—they break up—the blood becomes contaminated, and the phenomena of *embolism* supervene. Such dangerous phenomena may be looked for about the fourteenth to the twenty-first day.

The tissue of the darker portions appear tougher than in health, presenting nearly a uniform section; there is no appearance of granules, and the part sinks in water. Dr. Jenner has injected such morbid lungs, and found that occasionally the centre of the lobule is really the point at which the diseased action is first set up. The development of the new material appears to be very deficient; molecular granular matter and delicate minute cell-forms compose its structure; and the specific gravity of the part is greatly increased (1.040 or more). Its color is generally slate-gray or flesh-like; and the lesion is commonly limited by a vascular boundary, forming something like a distinct line of separation between comparatively healthy texture and local lesion.

Carnification occurs often in considerable portions of the lung (WALSHE). The general debility of the typhoid state seems to favor the occurrence of pulmonary collapse—a condition which must not be confounded with the hepatization of pneumonia. The tracheal glands, and those of the bronchial mucous membrane, are also affected.

There is generally a great tendency to ulceration of mucous membranes, in typhoid cases,—ulceration of the pharynx occurs in about *one-third* of the cases; of the larynx and œsophagus in one out of every fifteen cases; and the mucous membrane of the colon becomes implicated in seven out of twenty cases. Louis found the colon affected by the second week in two out of fourteen cases, by the third week in six out of twenty-three cases, by the fourth week in four out of fifteen cases, and between the fifth and tenth week in one out of two cases. There is also a tendency to pericæcal abscess, preceded by the phenomena of the morbid state known by the name of *perityphlitis*, or inflammation of the areolar connective tissue round the *caput cæcum*.

Growth of Tubercle during Typhoid Fever.—There is still another pulmonary condition which frequently occurs in *typhoid fever*, and

which may either complicate the progress of the case or come on subsequently to the fever. It is the development of tubercle. Usually when recovery takes place from *typhoid* fever it is complete; but in some cases, especially where there is hereditary predisposition, an impetus or tendency seems to be given to the development of tubercles in the lungs. If the physical signs of bronchitis continue beyond the thirtieth day, or fourth week, combined with hurried and difficult breathing, and with the signs and symptoms of great irritation of the lungs, then there are good grounds for suspecting that the deposition of tubercle has commenced in the lungs. Dr. Stokes gives two sets of cases in which this deposit takes place. In one set a great quantity of tuberculous matter seems to be formed during the existence of the fever; and although, sometimes, such an occurrence may not have been suspected, yet the expectoration of pulmonary calculi, at periods of different duration after the convalescence, furnish strong proofs that such a lesion had taken place. In other cases, again, the cure may be effected through absorption, or by suppuration of the minute tuberculous points over the mucous surface of the bronchia. A doubtful convalescence, a quick pulse, and a hectic state, suggest such a state of things, especially when combined with persistent bronchitis.

Erysipelas, phlebitis, parotitis, and such like local inflammations, are not uncommon in typhoid fever. Such lesions may be excited by cold simply; but the absorption into the blood of putrid substances, from the ulcerating patches of Peyer or other diseased parts, may be usually, and probably correctly, considered to be the cause of most of the secondary inflammations already noticed to occur in typhoid, enteric, or intestinal fever. Dr. Parkes considers it probable, however, that deficient urinary excretion may have a share in their production (Parkes *On the Urine*, p. 254).

Such are the more obvious secondary affections which may develop themselves during the progress of *typhoid* or *enteric fever*, and the derangements which these give rise to constitute new phenomena in its course. In some severe cases, however, the fever may destroy the patient in a few days, without leaving a trace of organic lesion in any part of the body.

These secondary affections just noticed all arise after the fever has existed some time; and it appears now to be pretty well established that the intestinal lesion at least is a special growth, which, in cases of recovery, follows first a progressive or developmental course, and afterwards retrogrades; just as in variola we first observe the development and maturation of the pustule, and subsequently its disappearance. The same may be said of the other local lesions in typhoid fever, although the existence of a special growth is not yet so fully established in the case of the thoracic and cerebral lesions, or in the parenchymatous, as compared with the mucous structures of the intestine; still, it is believed by some that an action more or less analogous to that which occurs in the *glands of Peyer* and the *minute solitary closed vesicles of the ileum*, occurs also in all the secondary lesions of typhoid fever in other parts (DR. STOKES). Specific characters of the elements composing the growth cannot be

shown to the eye even by microscopic examination. There is nothing in it to distinguish it from other elementary morbid products which are deficient in the power of organization. Dr. Stokes gives the best illustration of its vital specific attributes, in the absence of any *physical* specific character. He says, if two specimens of pus be taken, one from a pustule of variola, the other from an ordinary ulcer, although they may appear similar, they have separate and different *vital* characters. So has the poison of typhoid fever a specific *vital* attribute peculiar to itself.

The nature of this so-called "typhoid deposit" has been the subject of much discussion. It is a new growth rather than a deposit; and is, in the first instance, confined to the gland-elements, and seems really to consist in a directly continuous development of the pre-existing cell or germinal elements of the diseased glands. Eventually it pervades the submucous areolar tissue as a yellowish-white substance, deposited or infiltrated in a layer beneath the gland-tissue. Professor John Goodsir, in his descriptions of the morbid anatomy of the cases he dissected at Anstruther, in Fifeshire, was the first to point out that the growth was in the first instance confined to the interior of the closed vesicles, which become much distended thereby. They ultimately burst, and discharge their contents either into the cavity of the intestine, or into the submucous tissue, if the vesicles rupture at the base, as in the severe and unfavorable cases; and if the vesicle is completely destroyed and falls, or if many of them do so, a number of little pits are left, which correspond to the sites of the vesicles.

The new growth has no specific structure to distinguish it from other morbid growths (WEDL, VIRCHOW, and others); and although a specific "typhous cell" has been described and figured by Gruby, Vogel, Bennett, and others, its existence is not proven.

The development of the new growth in the glands begins like a simple hypertrophy of the gland-cells. Nuclei and cells exist in great abundance, which afterward degenerate into the abnormal diseased product of the typhoid masses. No forms arise capable of sustained existence; but a directly continuous development from pre-existing cell or germinal elements of the glands, the follicles, and the connective tissue, furnishes the material of the mass (VIRCHOW). An increase in the colorless corpuscles takes place in the blood, and deposit of pigment in the ganglia of the sympathetic nerve-system (VIRCHOW). When the gland vesicles burst, the exuberance of new growth gives a fungating appearance to the part; and when the rose-red tumor is cut into, a milk-like turbid juice exudes; and in this juice many new-formed elements may be seen, consisting of cells mostly oval or angular, with single eccentric nuclei—sometimes with many nuclei. The cell-contents are finely granular, and fat-globules may conceal the nucleus. The growth must be examined before ulceration commences; for, as softening advances, a viscid fluid with a bloody tinge, containing fine molecular elements, is all that remains, with decaying blood and blood-crystals.

Circumstances under which Death may ensue in Cases of Typhoid Fever: 1. By poisoning of the blood generally, as indicated by many

symptoms which typhoid fever has in common with typhus fever, cholera, small-pox, dysentery, scarlet fever, diphtheria, ichorrhæmia. The intensity of the fever (measured by the thermometer) is generally great in those cases, and the fatal event occurs either at a very early period of the fever, associated with cerebral congestion, or it may occur later, when it may be supposed that the danger is past. This is sometimes termed the secondary poisoning of the blood (septicæmia), and is most likely due to the ulcerated intestines, with the bowels perhaps on the verge of perforation. The pulse becomes rapid and small; cold, clammy sweats appear; and the body begins, even in life, to exhale a putrid odor. In cases where the blood is so gravely implicated, gas has been observed to become developed during life, and has been detected in the veins at the root of the neck for some minutes before death (CLOSS, FRANK, and JEFFREY MARSTON in *Med. Times*, Feb. 7, 1857).

2. By implication of excretory organs at an early period—for example, the kidney, as denoted by albuminuria, or by bloody urine—conditions which tend to aggravate the blood poisoning.

3. By congestions of important organs—for example, the lungs and the brain, in consequence of poisoned blood; and which congestions are still further brought about by the circulation, in the bloodvessels, of putrid juices, or of the substance of fibrinous debris of clots in a granular condition, having formed as plugs in the varicose veins surrounding the sloughs and ulcers of the intestines.

4. By hemorrhage from the bowels during the separation of the gland-sloughs.

5. By exhaustion from profuse diarrhœa in cases where the catarrh of the mucous membrane has been excessive.

6. By peritonitis, with or without perforation of the intestines. There are two periods in the course of the fever when perforation is apt to take place. The first period is during the separation of the sloughs, about the end of the second and throughout the third week. The second period is during protracted convalescence, with atrophy of the intestine already described, and when the ulcers are in a weak atonic state, the result of intense protracted fever and profuse catarrh.

7. By peritonitis subsequent on suppuration of the large mesenteric glands, and rupture of their inclosing capsule (JENNER); or from the bursting of softened new growth from the spleen into the peritoneum (ROBERTSON, JENNER); or from ulceration of the gall-bladder. The average mortality among cases of typhoid fever appears to be about 1 in $5\frac{1}{2}$ to 1 in 6. It is considerably less in autumn than in spring; and is least of all in winter. It tends to be greater among males than females; and the average age of fatal cases appears to be about 23.5. The mortality increases to a small extent as life advances. The disease in certain places seems never to be absent, and is invariably most prevalent during autumn, at the time that diarrhœa is most common; and it has been observed to be especially prevalent in seasons remarkable for their high temperature (MURCHISON).

[**Prognosis.**—The prognosis in typhoid fever should be always very

guarded, for, besides being a very severe disorder, intestinal perforation may happen in the mildest cases, and where, from the absence of well-marked symptoms, it has been difficult to make out a diagnosis, as well as in the course of severe attacks, and also during convalescence. When it occurs, the patient complains first of a feeling of unusual warmth in the abdomen, followed by sharp pain, heightened by pressure—the weight of bedclothes, poultices, and fomentations, being intolerable—which quickly extends over the whole abdomen. This is soon followed by nausea, and instant and obstinate vomiting of greenish matter, hiccup, a pale, shrunk, anxious face, a rapid, small pulse, tympany, constipation, retention of urine, and cold, clammy sweat over the whole body. Shortly before death all the violent symptoms usually cease.

The younger the subject the better the chances of recovery. After forty, typhoid fever is a very fatal disorder. It is more fatal when it attacks robust than feeble persons. A sudden remission, followed by an exacerbation of the symptoms, is, says Dr. Chomel, invariably mortal. With respect to the prognostic value of particular symptoms, it may be said that parotitis is a very unfavorable symptom; Dr. Trousseau has never seen a patient recover with this complication. Deafness, when limited to one ear, should cause a guarded prognosis. Early somnolence is a very unfavorable symptom, as well as the persistent declaration of the patient that he is quite well. Trousseau agrees with Graves that intestinal hemorrhage is not to be looked on as, generally, an unfavorable symptom; indeed, he seems to regard it as useful, unless abundant and frequent, and accompanying marked adynamic and ataxic symptoms. Great rapidity of pulse, with feebleness or extinction of the first sound of the heart, or intermittence of the beats, or a persistently quick, small, and contracted pulse, are met with in fatal cases. Early active delirium is a bad symptom. Few pregnant women attacked with typhoid fever, who abort in the course of the disease, recover. (See Spinal Symptoms, p. 350.)

Diagnosis.—Although there is no one, nor even two or three, symptoms actually pathognomonic of typhoid fever, there are certain phenomena which are met with so constantly, and with such greater frequency in its course than in any other disorder, that in most cases, when fully developed, a certain and timely diagnosis can be made. When a febrile disorder of several days' duration, in a temperate climate, is attended with early and marked prostration of strength, persistent frontal headache, a puzzled and stupefied expression, epistaxis, diarrhoea, tympany, pain and gurgling on pressure in the right iliac region, an eruption of lenticular rose-colored papulae on the chest, belly, or back, sudamina, enlarged spleen, diffused bronchitis, a pungent heat of surface, with persistent increase of the natural temperature of the body every evening, there can be no doubt, with such a body of symptoms, of the nature of the disorder. But many of these phenomena may be absent; for in the mild or latent forms of the disease the objective symptoms are few, feebly marked, and unsatisfactory; and are but little noticed either by the physician or patient. In such cases the diagnosis is perplexing and difficult. Still, careful observation, and familiarity with the disorder, will usually enable the physician to decide positively, even in the early stage. The duration of the attack, under such circumstances, becomes an important element in the diagnosis. Regular daily increase of the body-temperature, at first in the evening, and afterwards, also, in the morning, accompanied with muscular debility, constant headache, loss of appetite, indifference, and a somewhat besotted look, in a person between eighteen and forty years of age, and particularly one who has recently come to a large town, or during the prevalence

of an epidemic of typhoid fever, and if, on careful examination, no local disease can be found, there will be good reason to suspect the presence of the disorder, even though the so-called distinctive symptoms are wanting. The absence of the prodromic symptoms special to the exanthemata, should prevent its being mistaken for them. The visceral inflammations of old persons are often latent, and accompanied with extreme debility; but a regard to the time of life, and careful physical exploration, will generally make the case clear. Granular meningitis in children, may be mistaken for typhoid fever. Besides the absence of the special symptoms of typhoid fever, as well as of the bronchial complication, in granular meningitis there are commonly constipation, frequent vomiting, retraction of the abdomen instead of tympany; and active delirium is an early symptom, followed by deep coma. The facial expression of the two disorders is totally different. Acute phthisis in some respects often resembles typhoid fever; but the great and increasing difficulty of respiration, the presence and site of the moist crepitant rhonchi, general lessened resonance of the chest, the peculiar hue of the surface, and, indeed, the absence of the really distinctive phenomena of typhoid fever, should hinder us from confounding two disorders, which really have but few common elements, and sufficiently distinguishing ones, to prevent an erroneous diagnosis.

The late Dr. Southey Warter has published some valuable thermometrical observations respecting the differential diagnosis of typhoid fever and typhus (*St. Bartholomew's Hospital Reports*, 1866). In typhus the rise of temperature pretty generally follows that of the pulse; but in typhoid fever he did not find this to be the case. While the pulse rarely is more than 120 before the 8th day, and this number is uncommon, the temperature reaches from 102.4° to 104.3° Fahr. In typhoid fever the highest afternoon temperatures are reached about the 4th day; the highest in typhus usually on the 7th, sometimes on the 8th, and rarely as late as the 11th or 12th days. Both in typhus and typhoid fever there is a singular tendency of the body-heat to rise every alternate day, especially in the odd days; in typhus rises are common, on the 13th and 16th days; in typhoid fever, on the 9th, 11th, and 15th.]

Origin and Propagation of Typhoid Fever.—It is now about thirty years since M. Bretonneau related to the French Academy of Medicine a series of cases in which the communication of this disease from person to person, and its modes of propagation in this way, were so evident as to admit of no reasonable doubt. Nevertheless, the conclusion arrived at has not been generally accepted by the profession, so that the communicability of typhoid fever has not met with general belief.

[Though, as a general rule, the contagious principle of typhoid fever is not very active, it is incontestable that the disease is communicable, and, as Dr. Trousseau remarks, the number of those who deny it lessens daily. Most of the French physicians living in the rural districts have always advocated its contagiousness, whilst those of Paris for a long time denied it. Dr. Nathan Smith, as far back as 1824, maintained that it was as contagious as small-pox or measles. In July, 1829, Dr. Bretonneau transmitted to the French Academy of Medicine a communication, in which he asserted the contagiousness of dothiententérie, as it prevailed in the country (*Archives Gén. de Méd.*, t. xxi, p. 57). Chomel, in his *Leçons de Clinique Médicale*, published in 1834, inclined to this opinion, though, as he acknowledges, it was contrary to the general sentiment. Gendron, of

Chateau-du-Loirs, has given in his memoir (*Jour. des Connaissances Méd.*) a number of indisputable instances. Louis, in his *Fièvre Typhoïde*, 1844, adopts the belief, and, in 1846, stated to the Academy that he had seen in Paris four cases, about which it was impossible for him to have a doubt. Contagion appeared to him to be especially manifest when the hospitals were overcrowded. The evidence of Forget, of Strasbourg, Moreau, Leuret, Mistler and Ruef, Jacques, Pulegnat, Patry, Lombard, Piedvache, Mayer, and a host of others, in favor of the communicability of typhoid fever, is irresistible. In 1857, Dr. Trousseau made a report to the French Academy of Medicine, in which the importation of typhoid fever into a district hitherto exempt, by individuals who had contracted it elsewhere, and its subsequent propagation from these centres, are satisfactorily made out. Murchison (*Continued Fevers of Great Britain*) has cited a number of examples. A very remarkable and conclusive one is related by Dr. Austin Flint (*Practice of Medicine*) as having come under his own observation. Dr. Bartholow (*loc. cit.*, p. 201), has mentioned some facts indicating the propagation of this fever by contagion. Wards which had been occupied by fever patients apparently became poisoned, and other cases admitted into them were liable to be attacked. This happened in Ward 1, National Hotel Hospital, Baltimore, so frequently, that the disuse of it by wounded became necessary. The same was observed at Lincoln Hospital, Washington. Dr. B. states that he has seen three instances in which patients contracted fever whilst lying alongside typhoid fever cases, and subjected to the emanations both from their persons and dejections.]

"In so vital a question" (writes Dr. Budd, whose views, as I have attempted to put them here, seem especially deserving of attention), "it is, I need scarcely say, of the highest importance that the actual truth should be generally known." And Dr. Watson, whose authority is undisputed, very justly remarks that, "If this fever be really contagious, it is not only erroneous but dangerous to hold the contrary opinion." "To what extent it is dangerous," continues Dr. Budd, "may be best measured by the fact, that in this country alone 20,000 persons die annually of this fever, and 140,000 more are laid prostrate by it." Thus vast is the field for the operation of preventive measures. And when the discovery and success of such measures must depend in a great degree on our insight into the real mode of propagation, it is at once seen what importance the question assumes. Dr. Budd has, I think, clearly demonstrated the following facts:

1. That typhoid fever shows a decided tendency to spread through a household, a school, a barrack, or a village, when it has been once introduced. The introduction and propagation of the fever in the Clergy Orphan School at St. John's Wood (*Lancet*, 15th Nov., 1856) is an instance in point. The first case was important, and the illness began ten days after arrival; within three weeks four more cases occurred; and then *nineteen* cases were simultaneously affected within thirty-six hours. The fever which prevailed in the Military School of La Flèche, in France, in 1826, is a no less instructive example. The outbreak commenced first with a few scattered cases, and the disorder spreading, the school was broken up earlier than usual, and the pupils sent home. But before this disruption could be carried into effect, sixty of the number were seized with typhoid

fever. Twenty-nine others carried the seeds of the fever with them, and were laid up by it at their own homes. Of these twenty-nine, it was ascertained that *as many as eight communicated the disorder to persons who were engaged in attendance upon them.*

In further illustration of the doctrine of communicability, Dr. Budd cites, with minute details, numerous examples in the village of "North Tawton," in which typhoid fever having once appeared in a household, it extended itself to one or more members of the family before it finally died away. During the prevalence of the fever in this village, it also so happened that three persons left the place after they had become diseased, and each of the three persons communicated the same disease to one or more of the persons by whom they were surrounded in the new neighborhood whither they went. While two of these men remained in the village of "North Tawton," they both lodged in a court having a single and a common privy in it, and next door to a house where typhoid fever was. In due course of time and events both took the disease. The third man was a friend who came to see one of the two men already sick. He assisted to raise his sick friend in bed, and while so employed was quite overpowered by the smell from the sick man's body. The sense of this pestilent smell harassed him for days. He felt very unwell from that time; and on the *tenth* day from the date of the event just noticed, he was seized with shivering, followed by the complete expression of an attack of typhoid fever, which was of long duration; and before he became convalescent two of his children were laid up with the same disease, as well as a brother, who lived at some distance, but who repeatedly visited him during his illness. Except in the houses of these men no fever existed in that part of the country. Further, most interesting and conclusive examples are given in Dr. Budd's admirable papers published in the *Lancet* of 9th July, 1859, which prove beyond question that typhoid (intestinal) fever is a most readily communicable disease. The facility for propagation, however, seems to be modified under particular circumstances of season, place, and habits of life; in other words, the propagation of the disease requires some special conditions, which may be said of the whole class of communicable diseases.

2. The disease having once occurred, the patient is protected from a second attack. The specific nature of the disease is thus also established, for the fever not only propagates itself, but propagates no other kind of fever—one case following another with the same constancy of specific type that small-pox follows small-pox or measles succeeds to measles.

3. There seems also to be a definite period in which the poison is latent after being communicated—a period of incubation, during which a definite interval elapses before the development of the fever begins. This period, according to Dr. Budd's experience, seems to be from about a week to ten or fourteen days. *The living human body, therefore, is the soil in which this specific poison breeds and multiplies; and that most specific of processes which constitutes the fever itself is the process by which the multiplication is effected.*

4. Like all other diseases of its kind, its origin is unknown; and the first case in the series of each of these outbreaks mentioned may either have been casual and imported, or it may have been due to a rekindling of some dormant germ left from a former similar attack.

5. The virulent part of the specific poison by which the disease is communicated is doubtless contained in the diarrhœal discharges which issue from the diseased and *exanthematous* bowel. These discharges drying up, the germs of disease are thus preserved as effectually as the crusts of small-pox preserve the virus of that disease. If, therefore, through atmospheric or other agencies, these germs obtain access to the living body, diarrhœa is brought about in the usual course of events, and the commencement of the disease thus communicated takes place. The discharges from the bowels of the person so infected, which are at once copious, numerous, and liquid, are thrown into the water-closet or the privy, and the drains, or systems of drains, become at once saturated with the specific poison of the disease in its most concentrated and virulent form.

Regarding, therefore, the drain, or system of drains, as a channel directly continuous with the diseased intestine of the infected person, the specific virus of typhoid fever may be propagated amongst healthy persons in one of three ways—namely:

(1.) By percolation through the soil into the wells which supply drinking-water to the inhabitants; (2.) By issuing through defects in the sewers into the air of the inhabited area; or, (3.) By exhalation through the aperture of small, ill-trapped water-closets or privies, which are at once the receptacles of the discharges from the sick and the daily resort of the healthy.

When the specific poison thus issues into the air, the *atmosphere generated is immeasurably* more likely to communicate the disease than that which immediately surrounds the fever patients. "There is reason to believe, however, that the duration of the period of incubation varies considerably, partly with the nature of the medium through which the specific poison finds admission to the living body, partly by reason of the conditions it meets with there, and still more so *in virtue of the greater or less intensity of the state of change* in which the poison itself may be at the moment of its reception." Hence the simultaneous seizure of a large number of persons within a definite interval after the occurrence of a single case points to some one or other of these modes of propagation. Such modes of propagation are thus the exact counterpart of what has been oftentimes observed (especially in schools) in the case of measles, scarlet fever, and, in former times, of small-pox also (Dr. W. Budd).

[The chief determining causes of typhoid fever in the United States Army during the war of the rebellion, where it prevailed to a great extent (21,977 cases and 5608 deaths in the first year), were, undoubtedly, animal emanations and privy gases. Dr. Bartholow's personal observations have convinced him that the last-named cause cannot be overestimated. He writes: "The cases of fever occurred most numerous where the diarrhœal discharges were most abundant, and were most exposed to

decomposition and dissemination by the air and water supply. Diarrhoea was common. Badly constructed sinks within a few yards of the camp were the rule, and not unfrequently no sinks were used, but the environs of the camp were converted into a general latrine. Privy odor was soon developed, and the drinking-water contaminated. When sufficient time had elapsed, typhoid fever declared itself amongst those who had not completed the seasoning process, or those who had acquired a special susceptibility to the poison." (*United States Sanitary Commission's Memoirs of the War of the Rebellion*, p. 200.)]

Preventive Measures, or Measures for Checking the Spread of Typhoid Fever.—The measures about to be specified have been made public through the writings of Dr. William Budd on this subject; and, *provided they are thoroughly and efficiently carried out*, it is believed that the recurrence of typhoid fever may be entirely prevented.

To enable us to judge of the extent of the infection to be destroyed, there are two elements to be taken into account,—*First*, The amount and duration of the intestinal discharge in each case; and, *Second*, The number of cases actually occurring. With regard to the first, Louis has found that the average duration of the alvine flux in cases of typhoid fever is *fifteen* days in mild cases and *twenty-six* days in severe cases. With regard to the second point—namely, the number of cases occurring—the Reports of the Registrar-General show that at least 100,000 to 150,000 cases of typhoid fever occur annually in England alone. In other words, "*every year in England more than 100,000 human intestines, diseased in the way already described, continue each, for the space of a fortnight or thereabouts, to discharge upon the ground floods of liquid charged with matters on which the specific poison of a communicable disease has set its most specific mark*" (BUDD).

The measures recommended for preventing the spread of this fever are founded on the power of chemical agents to destroy absolutely the material which contains or carries the specific virus of such communicable diseases. Assuming it, therefore, to be certain that the intestinal discharges in typhoid fever are the media of propagating the disease, it is no less certain that, by SUBJECTING THE DISCHARGES ON THEIR ISSUE FROM THE BODY TO THE ACTION OF POWERFUL DECOMPOSING CHEMICAL AGENTS, THEY MAY BE ENTIRELY DESTROYED OR DEPRIVED OF THEIR SPECIFIC VIRUS. He suggests the following details of procedure:

1. All discharges from the fever patient should be received on their issue from the body into vessels containing a concentrated solution of chloride of zinc.

2. Two ounces of a caustic solution of chloride of zinc should be put in the night-stool on each occasion before it is used by the fever patient.

3. All tainted bed or body linen should, immediately on its removal, be placed in water strongly impregnated with the same agent.

4. The water-closet should be flooded several times a day with a strong solution of chloride of zinc; and some chloride of lime should be also placed there to serve as a source of chlorine in the gaseous form.

5. So long as fever lasts, the water-closet should be used exclusively as a receptacle for the discharges from the sick.

The Privy Council have now made the principle of this method an integral part of their "general memorandum on proceedings advisable to be taken in places attacked or threatened by epidemic diseases," and which is given *in extenso* at page 227 of this volume.

Vague and untenable notions have been gathering round this subject, particularly in relation to the propagation of typhoid fever; and if once a disease of this kind is decidedly proved to be the result of a specific poison, and to be propagated in the way just described, "we cannot help entertaining a doubt," says Dr. Watson, "whether the disorder in question really ever has any other cause." Nevertheless, other causes are assigned to typhoid fever; which by some is even looked upon as a disease developed out of external conditions alone. In reasoning on the subject, it must be remembered at the outset that the dissemination of typhoid fever by a *specific poison* implies precisely what it implies in small-pox; and that it is provided for in the same way—namely, by the multiplication of a *specific poison* in the living but diseased body. Each of these specific poisons (and, as we have already seen, they are numerous) thus multiplies in the same way, and in the same remarkable medium, out of the same living organisms of the human frame; yet each of these several poisons sets up a series of changes which always issue in the reproduction of its own specific kind of disease, and no other. Small-pox gives rise to small-pox, scarlet fever to scarlet fever, measles to measles, and so on. Herein lies their *specificity*. Such being the doctrine attempted to be maintained in these pages, the theory of the spontaneous origin of typhoid fever, or of any other specific disease, must be in the same relative position as when it seeks to explain by such a principle the propagation of plants and animals. These—namely plants and animals—likewise at least two diseases—namely, *syphilis* and *small-pox*—are certainly now known to propagate only by the law of continuous succession, whatever may have been their primary source. But the hypothesis of spontaneous origin and indefinite propagation of typhoid fever has assumed a definite form of expression in the doctrine which attempts to teach that typhoid fever is often actually caused by the products of common putrefaction—a doctrine which has been cleverly embodied in the nomenclature of the subject by Dr. Murchison; and thus a degree of precision and permanence has been given to the opinions he has so ably advocated in a volume on the *Continued Fevers of Great Britain*—a volume which is unsurpassed for its erudition and its practical importance.

The term "pythogenetic fever," or fever "born of putrescence," is the name by which Dr. Murchison at once designates typhoid fever and theoretically implies its origin. He has thus rashly committed Science to a hypothesis of a highly doubtful nature. Without, however, doubting the fact that animal and vegetable substances in some states of decomposition have the power of inducing ill-health, and that there is now acknowledged to be a connection between putrid states of the air and the prevalence of pyæmia, erysipelas,

puerperal fever, and cholera, yet there does not seem to be sufficient evidence to show that any of these causes can *produce* a disease which is of so specific a nature as to be maintained and propagated by a specific poison generated in the body alone. Undoubtedly, the state of ill-health induced by the decomposing material of night-soil and the like, does produce *a state of the system favorable to the development*, not alone of typhoid fever, but of many other specific diseases, such as cholera, dysentery, yellow fever, and the like. This predisposition to such diseases seems to be exactly analogous to the preparation of a soil for seed. Dr. Carpenter, also, long ago, showed physiologically what observation has since confirmed—namely, that decaying animal material, especially night-soil, seems to be for some poisons (*e. g.*, cholera and yellow fever) great centres or foci, where the specific germs or poisons are able to multiply; and for the propagation of which “foulness of medium is indispensable.” An interesting question for inquiry is thus opened up as to whether the germs of typhoid fever, cholera, and the like, could be made experimentally to grow or increase upon or about organic matter, just as the germs or spores of many fungi are induced to grow in collections of manure (DR. LANKESTER). In such collections on the earth’s surface there is reason to believe that germs of diseases like cholera and typhoid fever, and yellow fever, may find a resting-place—that thus they are always extant somewhere—although it may be only now and then, when season and other conditions conspire, that they display their full power as epidemic diseases. As such, they seem to occur every now and then as “mysterious cycles,” the existence of which we admit, but do not understand. The experiments of Dr. Barker on cesspool air prove that long inhalation of an atmosphere charged with the gases evolved from decomposing organic matter is capable of producing a series of symptoms of the following character—namely, increased heat of the skin, thirst, irregular and feeble muscular contractions, and diarrhœa. These symptoms continue so long as the person is exposed to the influence of the foul air; but when the cause is removed, there is no continuance of symptoms, no recurrence nor remittency, but a tendency to recovery. No communicable disorder is induced. The poison of the foul air acts for the time as chloroform might act, and so soon as removed, recovery progresses.

The history of typhoid fever, whose leading features have been described in the previous pages, is wholly inexplicable upon the “pythogenetic theory” of Dr. Murchison. On the contrary, it is emphatically the history of a specific disease generating a specific poison, and propagating itself by it:

“*Mutatis mutandis*,” writes Dr. Budd, “it is the history of *small-pox*, it is the history of *scarlet fever*, it is the history of malignant cholera. In all these specific contagions we meet with these same alternations of slumber and activity; of widespread prevalence in one place, while other places hard by remain free; and finally, with the same successive invasion of neighboring places, in such wise that the reigning disorder—be it *small-pox*, measles, scarlet fever, intestinal fever, or malignant cholera—

often only begins to prevail in the new locality when it has already died out in the old.

"It is, in fact, in a general survey of this kind that we get the clearest view of the thread which really connects all these circumstances. There is plainly but one thing constant; that is to say, a specific morbid cause—a cause which is neither a permanent product of the soil, or air, or of particular seasons, but which is susceptible of transmission from place to place; which breeds as it goes, and then again dies out or becomes dormant, without leaving any sign to mark its track.

"There is only one thing of which these can be the characteristics; and that is the specific poison which is bred of the disease, and by which the disease propagates, and which, in common with the other specific poisons perpetuated by the same law, possesses all these properties.

"Thus, when we come to scrutinize closely this course of the fever, even in these broad relations, we are again brought to recognize that which we have already proved by direct evidence—namely, its essentially contagious nature. This is the master fact in its history—the fact which governs all the rest."

But whatever may be the view theoretically adopted regarding either the origin or the propagation of typhoid fever, it is satisfactory to know that, practically, medical officers can employ preventive measures which (to use the words of Dr. Lankester) "will cover the issues of both theories." These measures have been minutely described; and if they are universally carried into effect, it is not too much to expect that this fever might perhaps soon become extinct. At all events, with the facts before us, it is unwarrantable to permit the great bulk of what escapes from the diseased intestine of typhoid fever patients to be let loose upon society, into the cess-pool or sewer, or on the dung-heaps, in full possession of all their deadly power, without being first destroyed in the way that has been recommended.

"The grand fact is clear," writes Dr. Parkes, "that the occurrence of typhoid fever points unequivocally to defective removal of excreta, and that it is a disease altogether and easily preventible." Typhoid fever ought therefore soon to disappear from every return of disease, whether in military or in civil life.

[Professor Joseph Jones has, recently (*U. S. Sanitary Commission Medical Memoirs, l. c.*), very ably and strongly argued against the validity of the theory of Dr. Budd, and Professors Aitkin and Parkes, respecting the chief ætic factor of typhoid fever. The comparative immunity of dust-men, sewer cleansers and hunters, and privy cleansers, and all men engaged in removing the filth and offal of large cities, from the various forms of continued fever, militates, he thinks, against it. He makes also the following interesting statement: "Notwithstanding the crowded and filthy condition of the Confederate Military Prison and Hospital [Andersonville, Ga.], and notwithstanding the effluvia from the immense collections of abnormal human excrements, only 472 cases of typhoid fever were reported amongst over 40,000 men, with an average monthly mean of 21.120, during a period of six months. The cases of typhoid fever constituted only a little over one per cent. of the entire number of cases of disease entered upon the sick reports, and of the entire number of prisoners confined during six months at Andersonville" (p. 601).

Professor Jones further remarks: "If the contagious nature of typhoid fever be admitted, might not the relative greater proportion of persons liable to the disease in the country and villages, where it more seldom prevails, as well as the intimate relations and associations, and constant visiting amongst the entire population, and especially the collection together at stated periods in one or more houses of worship of a large proportion of the inhabitants, account for the apparent more extended action of the fever after its introduction into small towns and villages, without the necessity of resorting to the hypothesis of the propagation of the disease by the emanations of the cesspools? However, if the entire lesion of typhoid fever be the characteristic manifestation of the disease, corresponding to the eruption of small-pox, we must admit that the theory of Drs. Budd, Simon and Aitkin, has much plausibility, and even probability. According to this view the discharges from the bowels in typhoid fever might be regarded in the same light, as far as their contagious nature was concerned, as the matter formed upon the surface, and cast off in the form of scabs in small-pox. The admission of this theory does not at all overthrow the assertion that animal putrefaction does not generate under any circumstances a contagious fever, for in the case of the excrements voided in typhoid fever, the poison is the result of the actions going on in the living body, and is not the product of the decomposing excrements" (p. 603).]

Treatment of Enteric or Typhoid Fever.—The chief indications of treatment are to reduce temperature and subdue vascular excitement, if these be in excess; to restrain and moderate, but not to suppress or check, the diarrhœa; to stimulate the nervous system when necessary; to obtain a free action of the kidneys; and to influence the elimination of the morbid growth from the intestinal glands.

To accomplish the first of these indications the use of *digitalis* has been especially recommended by Wunderlich. He considers that it decidedly mitigates the febrile symptoms which are present in severe cases at the time when the ulcers begin to heal, and which often impede or prevent recovery. He advocates its use in the severe forms of the fever only, especially at a time when most danger is to be apprehended from the violence of the fever in the second week, when the evening temperature is at its highest (105° to 108° Fahr.), and when the remissions in the morning are slight; when the pulse is frequent, 110 to 120, or more. In mild cases it is superfluous. He finds that in the form of *infusion* it is easily absorbed by the intestines of patients suffering from fever; and, if given in a suitable dose, has most marked effects in subduing the rate of pulsation, and in reducing animal heat. Large doses of the infusion should be given without interruption until the full effect has been obtained,—

An infusion of fifteen or twenty grains of *digitalis* in eight or ten ounces of boiling distilled water may be consumed in twenty-four hours by adult patients.

It acts more rapidly on animal temperature than on the heart. For the first few days after its use the decrease of temperature is rather slight, but may afterwards become considerable; and after it

has been much diminished and again rises, it never attains its former excessive height. The full effects of the medicine are known to be brought about if the temperature is reduced to 2° or 3° Fahr. in the evening; and the action of the remedy does not continue beyond one day after its use has been discontinued. The diminution of the pulse is slight at first, and occurs in some cases on the second day after the remedy, but mostly on the third day, or even later; and on the fourth or fifth day after the medicine has been commenced, the rate of pulsation may be diminished by thirty or forty or fifty beats within from twelve to thirty-six hours. The pulse may continue to fall even below its normal velocity, and this reduction of pulsation may last for several weeks in succession. If the velocity of the pulse should decrease rapidly, the use of the digitalis must be discontinued at once (*Arch. der Heilk.*, 1862, p. 116).

[The tincture of the root of aconite, in small and frequently repeated doses, alone or combined with digitalis, will, when the vascular excitement is excessive, be found efficacious.]

Cold and tepid sponging [with vinegar and water, to which some aromatic spirit has been added], or the cold affusion, are remedies which Dr. Murchison considers deserving of further trial for reducing the pulse and temperature.

[Packing in the wet sheet would seem, from the experience of Flint and others, to reduce the heat of the surface, diminish the force and rapidity of the pulse, and calm the nervous symptoms. It should be used not later than the first week.]

The oppressive headache may be greatly relieved by cold to the head, [though often tepid, or even warm, applications will give more relief]; and in all severe cases the hair should be shaved off.

To restrain excessive diarrhœa, *lime-water mixed with milk* in equal parts, and taken as a drink, is found to be beneficial, agreeable, refreshing, and nourishing. It is clear that the diarrhœa ought not to be altogether checked. Professor Gairdner, of Glasgow, is in favor of the French practice of giving saline laxatives rather than astringents; also of the diligent use of enemata to unload the bowels from below, where anything like abdominal distension has occurred. These enemata may be simply of warm water, to which a little aniseed, [or tincture of rue,] is added; or the assafœtida enema may be given. On the propriety of restraining *excessive* diarrhœa there can be no question. Dr. Huss, of Stockholm, is of opinion that the diarrhœa during the first stage ought not to be arrested, but abated and mitigated only *if excessive*. If it is suddenly arrested, meteorism is produced, and pains in the intestines; or vomiting may supervene, with cerebral symptoms, and the febrile phenomena are increased. The diarrhœa is too copious or excessive if the evacuations exceed four or five a day, being of considerable quantity, and fluid. Such evacuations weaken the patient rapidly, and should be mitigated by mucilaginous drinks, such as rice-water, infusion of linseed, decoction of *althœa officinalis*, or *ipecacuanha* in

small and repeated doses, [or prepared chalk, with or without bismuth.] This latter remedy retards the peristaltic action of the intestines, and lessens the secretion from the mucous membrane. The dose must be regulated so as to avoid vomiting; and the feeling of nausea which is apt to follow the first dose soon disappears with continued use.

Dr. Murchison, on the other hand, agrees with the late Dr. Todd, who writes as follows: "Restrain diarrhœa and hemorrhage in typhoid fever, and when you have fairly locked up the bowels, keep them so. Patients will go for four or six days, or even longer, without suffering inconvenience from this state of constipation." Dr. Huss and Dr. Murchison speak highly of the benefits to be derived from the mineral acids—hydrochloric and sulphuric* especially. From fifteen to thirty minims of the dilute acids may be given every three or four hours; and with each dose Dr. Murchison recommends half a grain of quinine, as in the following prescription for an adult:

R. Acid. Sulph. dil., vel Acid. Hydrochlor. dil., ℥xx. ad. xxx.
 Quiniæ Sulph., gr. ½ ad. gr. j.
 Syrup. Aurantii, ʒss.
 Aquæ Carui, ad. ʒj.
 Fiat haustus, 3â vel 4â, horâ sumendus (MURCHISON).

He is of opinion that if there be more than two motions in the twenty-four hours, with marked prostration, that astringents should be had recourse to. A starch enema, containing from ten to twenty drops of laudanum, should be administered towards evening, and recourse may also be had to the following draught:

R. Acid. Sulph. Aromat., ℥xxx.
 Liq. Opii. Sedativ. (Battley), ℥ij.
 Aq. Menth. Pip., ʒj; *misce*.
 Fiat haustus, 4â vel 6â, quaque horâ sumendus (MURCHISON).

If the mineral acids are not tolerated by the stomach, acetate of lead is worthy of trial, in doses of two or three grains in solution every four or six hours, with or without an eighth of a grain of morphia (MURCHISON).

Alum dissolved in gum, to the amount of twenty-four grains in a day, which may be increased to one drachm, is best given in the

* [With a view of ascertaining any curative influence that might be exerted by sulphuric acid in continued fever, Dr. Irving W. Lyon instituted a comparison in the rate of mortality in the male fever ward at the Bellevue Hospital, New York, in cases treated with and without the acid, during periods of six months. He left out all cases where death took place within forty-eight hours after admission. From January 1st to July 1st, 1863, 70 cases of fever were treated without sulphuric acid, with 14 deaths, or 20 per centum. From July 1st, 1863, to January 1st, 1864, 78 cases were treated with sulphuric acid; there were 8 deaths, or 10.26 per centum. The general treatment in both series was essentially the same. The apparent reduction of mortality was one-half (*Am. Med. Times*, February, 1864). Magnus Huss, of Stockholm, very strongly recommends dilute phosphoric acid, in hourly doses of about ten minims.—EDITOR.]

form of *alum whey*, prepared by adding one drachm of alum to a pint of boiling milk, and then straining. Two ounces may be given after each motion of the bowels (FOUQUIER, MURCHISON).

A Seidlitz powder may sometimes check excessive diarrhœa, by altering the secretions, and is especially beneficial if there be much meteorism (TROUSSEAU, MURCHISON).

The question, therefore, of checking excessive, or encouraging moderate diarrhœa is one which requires the careful study of each particular case; and my experience in the treatment of typhoid fever leads me to recommend rather the restraining of excessive diarrhœa than either encouraging the action of the bowels or locking them up for days. The French practice of giving saline laxatives is not a safe one; and if the action of the bowels is deemed desirable, enemata will be found sufficient. Nitrate of silver, in doses of one to three grains, made into a pill, and taken every six or eight hours; or sulphate of copper, in doses of a quarter of a grain similarly given, are remedies most useful in the diarrhœa connected with atonic ulcers, after the fourth week of the disease (BELL, MURCHISON).

[Should there be constipation, either initial or intercurrent, small doses of castor oil, and tepid enemata of infusion of chamomile, will be found preferable to saline laxatives.]

If meteorism or tympanitis prevails, from the accumulation of air in the colon, it may sometimes be relieved by the passage of a long stomach-pump tube by the anus, as far up the colon as it can be made to go easily.

With regard to stimulation of the nervous system, it is not decided how far alcohol is beneficial. The fever eventually is attended with much exhaustion, and is often protracted, yet typhoid fever does not seem to bear stimulation so well as typhus. The tendency to prostration is the only indication for its use; but stimulation must not be persevered in if the pulse is quickened by its use, the hectic flush made more manifest, the tongue made drier, or if delirium supervene. Food and sustenance are the real preventives of delirium, and the best stimulants to the nervous system when necessary. With regard to the maintenance of free action from the kidneys, it is to be observed that so long as the *excretion* of urea and uric acid is abundant, no diuretics are necessary; but whenever the amount of the solids falls greatly (which can be known at once with sufficient accuracy by a comparison of the urine passed in twenty-four hours with the specific gravity), means must be taken at once to increase, if possible, the urinary elimination. The *warm bath*, with repeated small doses of the *alkaline carbonates*, or of the *nitrate* or *bitartrate* of *potash*, will often effect an increased elimination (PARKES, *l. c.*).

To accomplish the last indication—namely, to influence the elimination from the intestinal glands, by direct local action on the intestinal membrane—Wunderlich especially advocates the use of *calomel*, if it can be given before the ninth or tenth day. Dr. Parkes is also of opinion that it is extremely useful at this period. The

late Dr. Anthony Todd Thomson used to give it; and, from the observation of many cases under the care of this physician, as well as from his own experience, Dr. Parkes considers that *calomel* is a medicine to be strongly recommended in typhoid fever. But it must not be given later than the tenth or eleventh day, and at no time in large doses.

One or two grains twice a day is enough, although Wunderlich gives one to five grains twice daily; but five grains is considered by Dr. Parkes to be too large a dose.

Dr. Wood, of Philadelphia, bears testimony also to the benefit to be derived from mercury about the seventh or ninth day of the fever. He believes "it tends in some degree to arrest the progress of the disease in the glands of Peyer, and to promote resolution of the inflamed patches. He prefers minute doses of the blue pill mass—a grain every two hours—till the mouth is slightly affected, associated with small doses of *ipecacuanha*, when the stomach is not irritable. The beneficial effect of this combination is shown by the tongue becoming moist, the skin relaxed, and the symptoms generally being ameliorated. Dr. Wood recommends twelve grains of *blue pill mass* to be combined with two grains of *ipecacuanha powder*, with two grains of *opium powder*; and the whole being divided into twelve pills, one may be taken every hour, or every hour and a half, or every two hours (*Practice of Medicine*, vol. i, p. 345, 4th edition).

Calomel is, however, contraindicated if the diarrhœa is excessive, or if there should be excessive pains in the bowels, with early and violent meteorism. It is also not proper to be given if the condition of the patient is anæmic, or if there is a decided hemorrhagic diathesis. It is most useful as a restorative of the intestinal functions in cases where the tongue is dry and coated, where thirst is absent, and when the urine is cloudy and of low specific gravity. If the first dose is vomited, the administration must be repeated. *Calomel* has no direct effect on the pulsation or respiration, nor on the cerebral functions; but its beneficial influence is very decidedly appreciable by the modifications of temperature which it induces, and which have been already noticed at page 360.

No general rules can be laid down to guide the treatment of the intercurrent phenomena or accidents of the disease. Combinations of remedies must be adapted to correct the several functions which may be simultaneously deranged, so that treatment must be varied according to the functions mainly implicated or suspended, and to the degree of their affection.

Abdominal pains and meteorism may be relieved by mustard poultices, or turpentine *stupes* may be applied, followed by simple hot water fomentations. After these remedies have been used, cold water compresses over the abdomen tend to lessen the tension and the gurgling in the intestines, and to diminish the tenderness on pressure. They counteract the inclination to meteorism, and lessen excessive diarrhœa. Dr. Huss believes also that the ulcerations in the ileum are prevented from spreading; and that perforations of the intestine have been of much rarer occurrence since he com-

menced to use these compresses. The compress, after being soaked in warm, but not hot water, is well wrung, and applied so as to cover the whole abdomen; and it must be changed two or three times a day, according to its tendency to dry. The compress is composed of four to eight double folds of coarse linen, and is to be laid over the whole abdomen, and afterwards overlaid with a cover of oiled skin or India-rubber stuff, to prevent too rapid evaporation. The compress should fit as closely as possible, and care must be taken that it be not displaced, otherwise air enters between the skin and the compress, so that cold, instead of a moist heat is produced. These compresses may remain untouched as long as they are moist and warm; and they may be employed on the chest as well as on the abdomen, should capillary catarrh or pneumonia supervene.

Hemorrhage from the bowels, partly fluid and partly in clots, evacuated with the stools, is a symptom of grave import; and the urgent aim of treatment is to arrest the bleeding. Huss found the *sugar of lead* most serviceable, administered by the mouth, and also by clysters. By the mouth, two grains may be given every half-hour, and even every quarter of an hour; and it is best given dissolved. Twenty-four grains of *crystallized acetate of lead* being dissolved in one drachm of *dilute acetic acid*, to which six ounces of *distilled water* is added, a tablespoonful of this solution may be given every half-hour. At the same time a clyster may be administered, composed as follows: Ten to fifteen grains of *acetate of lead* are to be dissolved in four ounces of *distilled water* (warm), to which twenty or thirty drops of tincture of opium may be added; and the administration of such a clyster may be repeated in four or six hours if required. Pieces of ice may also be swallowed now and then; and even crushed ice may be applied, inclosed in a bladder, over the abdomen.

[A combination of sulphuric acid and syrup of rhatany is praised by Trousseau. Five drops of the Elixir Halleri in camphor-water, frequently repeated, acts well.]

If bleeding from the nose is not arrested by the use of vinegar and cold water injected up the nostrils, nor by the use of cold water compresses applied to the nose, plugging by the posterior nares must be had recourse to.

Turpentine is a most valuable remedy in hemorrhages, and in the stage of ulceration. It was originally recommended by Dr. Graves, and is highly spoken of by Drs. Huss, Wood, and Murchison. It may be given in all cases where the tongue is dry, and when, "instead of cleaning gradually from the edges and tip, it often parts with its fur quickly, and in large flakes—generally, first, from the middle or back part of the surface, which is smooth and glossy, as if deprived of its papillæ. There is also generally an increase of the tympanitis, and the ulceration of the ileum seems to be attended with great dryness of the tongue." Under these circumstances Dr. Wood gives the oil of turpentine in doses of five to twenty drops every hour, or every two hours. It is best administered in an

emulsion with gum arabic, loaf sugar, water (WOOD), or in an emulsion with the yolk of an egg and honey or mucilage (HUSS). Amelioration of the symptoms may be observed in twenty-four or forty-eight hours—the tongue becoming more moist, and covered with a white fur—distension of the abdomen ceases to progress, and after a time diminishes. The use of the oil should be continued under these circumstances; but the dose should be gradually diminished.*

[The chlorate of potash, in doses of five grains, may be given in camphor-water or weak bitter infusions, every two hours. Under its use the tongue often becomes clean and moist (GARNETT, HUNT, COPLAND, WATSON). Chlorinated soda—ten to fifteen drops in camphor julep—is highly praised by Copland, Chomel, and Graves.]

Tonics and stimulants may be absolutely essential on account of debility attending the advanced stage of the disease, generally about the third week. When the pulse is slow and feeble, the skin cool, the tongue and teeth incrustated with dark sordes, at an advanced period of the fever, then stimulants are obviously necessary. But even when the pulse is feeble, but yet frequent, and the skin hot, stimulants are even then known to be of service; but it is necessary to administer them with great caution, and to watch the effects constantly and closely. If their use is found to augment the heat of the skin, and to increase the frequency of the pulse, and to aggravate the delirium or stupor, it is then necessary to suspend their use. They are known to be doing good service, however, if they lessen the frequency of the pulse, and increase its fulness and strength; if the skin becomes cool and moist, and if the delirium is subdued or moderated; and especially if refreshing sleep be procured. Dr. Wood recommends the use of wine whey, prepared by adding *one quart of good sherry wine to two quarts of boiling milk*, and straining after coagulation. Of this a tablespoonful or more may be given every hour or every two hours. If the strength is greatly reduced, it may be necessary to give pure wine or brandy; or even sulphuric or chloric ether in cases of great prostration.

[If a patient with typhoid fever is properly nourished from the outset of the attack, alcoholic stimulants will not be necessary in a large number of cases. They are greatly overused. When, in spite of the early and regular administration of food, there is great prostration, or ataxic phenomena come on, stimulants should be at once prescribed; sound sherry wine is the best form for their administration, or, where diarrhoea is excessive, a pure Port, or Tarragona, wine; they are best given with milk, eggs, or broth. Iced champagne is often very grateful and happy in its effects, particularly where there is obstinate inappetency, or gastric irritability. Brandy is preferable to whiskey. It has been pro-

* [The value of turpentine in typhoid fever is very doubtful. After a good deal of experience with the turpentine treatment, the writer has never seen any result which could be fairly attributable to it, except disordering the stomach. Dr. Gerhard's testimony is to same point; he says: "I cannot think it of much value . . . in ordinary cases it is perfectly nugatory." (*Pennsylvania Hospital Reports*, 1868.)—EDITOR.]

posed to deduce from the cardiac phenomena a rule to regulate the administration of stimulants in typhoid fever—the diminished cardiac impulse, and the feebleness, or extinction, of the first sound, being a direct and important indication for their use (STOKES, PENNOCK). May not the experiments of Claude Bernard, Marey, and de Barrel de Pontevès warrant us in regarding the therapeutic action of alcohol and its preparations to be more directly upon the sympathetic than upon the cerebral system, thus lessening the periphero-vasal tension, and increasing the cardiac impulsion? and is not this view more in accordance with the observed phenomena than the vague generality of a hinderer of metamorphosis, or a retarder of organic combustion? Dr. Warter thinks (*l. c.*) that the body-temperature will best indicate the proper treatment of delirium. If the thermometer marks a low temperature, wine and food may be given with benefit.]

Opium is also a useful stimulant. It may be given when the pulse is not full nor strong, and when cerebral symptoms do not exist. In the later stages it may be given in doses of half a grain, or a grain, every four, six, or eight hours. It is known to be acting beneficially when it promotes sleep, subdues nervous excitement, and induces gentle perspiration.

[Opium is a valuable remedy for the nervous symptoms, but its employment is a point of great nicety, and requires caution and discrimination. In delicate and nervous patients, or such as have largely used spirituous and vinous liquors, or where nervous energy is prostrated, and there is little general or local vascular excitement, with a cool, moist skin, in such, when low delirium, restlessness, wakefulness, and twitching come on, opium will do good; it should be at first given in small doses, and its effects carefully watched. Sir Henry Holland suggests that the condition of the pupil may serve as a guide in some doubtful cases—where it is contracted, opium being contra-indicated. Dr. Law, of Dublin, and afterwards, Dr. Graves, speak highly of a combination of laudanum and tartar emetic in controlling cerebral symptoms. Ataxic symptoms are, sometimes, quickly checked by camphor, and it will often bring on general composure and easy sleep.]

Carbonate of ammonia is objectionable, as it may irritate the bowels and increase the diarrhœa.

The diet is of the utmost importance to be attended to in cases of typhoid fever from the very commencement of the disease.

[We should hinder autophagism, resulting from the rapid and excessive destructive metamorphosis in all the tissues. Protracted abstinence is not only a common cause of death in low fevers, but of many of the adynamic, cerebral, ataxic and gastric symptoms. If there is one point in the treatment of typhoid fever to be insisted on, it is the administration of proper nourishment; indifference or repugnance to food on the part of the patient, should not interfere with this chief remedy, the appetite often coming on after a day or two of persistent nourishment.]

It ought then to be both food and drink combined, in the form of a light nutritious liquid. Barley-water, rice-water, toast and water, thickened more or less with solutions of tapioca, sago, arrow-root, the juice of sweet fruits, or the very soft pulp of fruits, or the

pure jelly of ripe fruits; but fruit in its crude state is to be strictly withheld. It is necessary, as a rule, to give food at certain intervals and in certain quantities. A wineglassful should be given at least every two or three hours [through the day and night,] according to the state of digestion and the demands upon the strength of the patient. It may be that the patient is unable to swallow, from the dry and shrivelled state of his tongue. Before offering him food or drink, therefore, the nurse should put a teaspoonful of lemon-juice and water into his mouth. She must then wait a minute or so, until the fur upon the tongue and mouth is softened and moist, after which the patient will often drink or take his food with ease. Milk in small quantities, [two or three ounces, to which a little lime-water, or bicarbonate of soda, or Vichy water, may be added, if there is much acidity of stomach, or cream, or a raw egg beaten up with water and sugar,] frequently repeated, will be found an excellent diet; and animal broths and jellies may ultimately be given. The *extractum carnis*, as prepared by Liebig, is a most valuable nutriment for typhoid fever patients.

[The patient should be placed under the most favorable hygienic conditions; he should be withdrawn from all disturbing or depressing influences, and perfect quiet of mind and body enjoined. Free and abundant ventilation and strict cleanliness of the apartment are indispensable, together with light bedding, which should be changed daily. The excreta must be removed immediately, and disinfectants and deodorizers frequently employed. Those parts of the body that may be exposed to pressure are to be daily examined, and if found reddened, they should be gently rubbed, and protected by some artificial cuticle, and a water- or air-cushion used. The state of the bladder should be ascertained frequently, and when necessary, the water ought to be drawn off.]

The utmost caution is necessary as to diet and aperients during convalescence; *first*, as to opening the bowels, castor oil or simple enemata are the only means which should be resorted to; *secondly*, as to diet, no flesh meat should be allowed till at least seven days after all the febrile phenomena have passed away, and the food should be as free as possible of excrementitious matter, [and at first should be given well hashed or minced.] Malt liquors should not be taken *before* food.

[During the whole of convalescence great care and vigilance are required, and the patient must be closely watched. Exposure to cold, or any fatigue or mental excitement, are to be strictly avoided. The quantity of food allowed must be rigorously within the capacity of the digestive function, otherwise there will be risk of gastro-intestinal troubles—inappetency, nausea, vomiting, gastric pain, tympany, diarrhoea, and intestinal perforation; or of evening fever exacerbations; or of a relapse. Bear constantly in mind the yet unhealed ulcers of the small intestine; strengthen the digestive organs; and be careful to do nothing that may weaken them. Most of the consecutive disorders of convalescence will gradually disappear as the waste of the system is healthily repaired. Paralysis, mania, dropsy, aphasia, if existing, cease by degrees as the strength returns. Wine, and the vegetable and mineral tonics are gen-

erally necessary during convalescence. Obstinate wakefulness yields to opium and wine. A cold infusion of the *Prunus Virginiana* is a valuable adjuvant when the pulse is persistently rapid.]

TYPHUS* FEVER.

LATIN, *Febris typhus*; FRENCH, *Typhus*; GERMAN, *Exanthematischer Typhus*—Syn., *Fleckfieber*; ITALIAN, *Tifo*.

Definition.—A continued fever, attended with sluggishness of intellect and confusion of thought, followed and accompanied by an eruption on the skin, of a rubeoloid character, appearing generally from the fifth to the eighth day, at first slightly elevated, and disappearing on pressure, but after the second day persistent, and remaining persistent for eleven or twelve days. Languor and weariness, prominent from the first, gradually pass into stupidity, oblivion, and complete prostration; which, in still more extreme cases, pass into somnolence, stupor, and sometimes coma, when prostration becomes profound. The disease may terminate favorably from the thirteenth to the seventeenth day, the average duration of the attack being about twenty-one days. If the disease proves fatal, it is generally between the twelfth and the twentieth day, leaving no specific lesion beyond hyperæmia, softening of the heart and contractile fibre-structures, and atrophy of the brain.

Historical Notice.—The first authentic accounts of typhus fever are to be found in the early British chronicles. It is described as having spread in our courts of justice, giving rise to what was termed "the black assizes." The last black assizes happened at the sessions of the Old Bailey in 1756, when the lord mayor, two of the judges, and several eminent persons died, infected by the prisoners. This fever has had many popular appellations, having been known as the *jail fever*, *hospital fever*, *ship fever*, *putrid fever*, *brain fever*, *bilious fever*, *spotted fever*, *petechial fever*, *camp fever*. We are indebted, however, to Pringle and to Fordyce for having shown that these supposed different fevers are identically the same, and have no such essential differences as to constitute them distinct genera.

Typhus fever is the grand scourge of armies in temperate climates, just as cholera and yellow fever have been the destructive agents in

* As the term typhus is very variously used, and sometimes vaguely, it is necessary to state precisely the meaning of the word. This cannot be more clearly, distinctly and concisely expressed than in the words of Dr. Wood.

The disease now defined is sometimes called *typhus*, and sometimes *typhus fever*. In the first instance the term is used "substantively, in the latter adjectively, just as we say ship fever, jail fever, &c. But a state of the system, identical or closely analogous with that which characterizes *typhus* fever, is frequently met with in other febrile diseases, as a mere incidental accompaniment. To this morbid state the epithet *typhous* or *typhoid* is applied, the latter being preferred to the former when it is wished to imply resemblance only, and not sameness or identity. Thus we speak of a typhous or a typhoid condition of remittent fever, yellow fever, small-pox, measles, pneumonia, dysentery, or, with greater brevity, *typhous pneumonia*, *typhous dysentery*. This latter phraseology, however, generally implies a more thorough incorporation of the *typhoid element* with the principal affection than *typhoid pneumonia* or *typhoid dysentery*, which merely implies a resemblance to the typhous state occurring in these diseases.

the tropical wars (Parkes "On the Causes of Sickness in English Wars," *Journal of Royal United Service Institution*, vol. vi). Wherever men are closely crowded together in ill-ventilated, unwholesome dwellings, typhus is sure to appear. It has often passed from the army to the civil population, and has thus dispeopled towns, and even great districts of country. But its ravages in the English army have never been comparable to those which have occurred in foreign forces, as the statements of Murchison and Parkes fully demonstrate: "In the year 1489 no fewer than 17,000 of the troops of Ferdinand, then besieging Granada, were destroyed by a spotted fever, to which the Spaniards applied the same name that they afterwards gave to typhus. In 1552 a petechial fever devastated the army of the Emperor Charles V during the siege of Metz. In 1556 the notorious '*Morbus Hungaricus*' appeared in Hungary in the army of Maximilian II, and thence spread over the whole of Europe" (MURCHISON, *l. c.*, p. 21). "In 1620 the Bavarian army in a few months lost in Bohemia not less than 20,000 men from spotted typhus; and the disease, being carried into other parts of Germany, obtained the name of 'the Bohemian disease.' In 1628 and 1632 the Swedish army under Gustavus Adolphus carried typhus into Northern Germany, and the population was so destroyed that, fifty or sixty years later, villages were left without inhabitants" (PARKES, *l. c.*). In the spring of 1643, while the Earl of Essex was besieging the town of Reading, this disease broke out in the army of the Parliamentary General, and in the garrison commanded by Charles I: it was communicated to the inhabitants of the surrounding country, and proved very fatal (MURCHISON, *l. c.*). The wars of Louis XIV were always followed by this disease, and the losses of the French army were enormous (PARKES). In 1799-1800 an epidemic of typhus occurred at Genoa, when the garrison was besieged by the French, and half famished; and the French army, during their retreat from Italy, communicated fever to the inhabitants of fifteen towns and villages where they halted on the route (FODERE). It was during the first fifteen years of the present century that the greatest ravages of typhus have been recorded, especially in the armies of Napoleon, and among the population of the countries which were the seat of war. It always became developed under circumstances of misery and privation, and was particularly prevalent and fatal among the inhabitants of besieged cities—as, for example, Saragossa and Torgau, Dantzic and Wilna, in 1803—and which told with such awful severity upon the famished French troops during the retreat from Moscow in 1812 and 1813 (MURCHISON). When Sir John Moore's army landed from Corunna, typhus became epidemic in the military hospitals in the south of England (Cheyne, *Dub. Hosp. Report*, vol. ii, p. 3). In May, 1812, the Bavarian army serving among the French numbered 28,000 men; in February, 1813, there were only 2250 men under arms. The great destroyer was typhus. In August, 1813, the first Prussian army consisted of 37,728 fighting men, having lost 16,000 men by the sword, and 10,000 men by disease, almost entirely typhus. In Mayence alone, of 60,000 French troops composing the garrison in 1813-14, there died of typhus in six months 25,000 men (MUR-

CHISON, p. 224). The last great ravages of typhus in armies which attracted public attention were those which occurred in the French and Russian armies in the Crimea during and after the Capture of Sebastopol. Typhus had prevailed in the winter of 1854-55 amongst both the English and French troops; but in the following winter it was mainly confined to the French and Russian armies. In the spring of 1856 it was computed that more than 17,000 men of the French forces perished in less than three months; and the highest authority stated that the safety of the whole French army was endangered by the outbreak (PARKES and MURCHISON).

According to Dr. Parkes, typhus fever occupies the fourth place among the causes which have produced disease in the British army.*

[During the war of the rebellion there was perhaps entire immunity from typhus in both the United States and Confederate armies. Though 1723 cases, and 572 deaths, are found in the returns of the Surgeon General's office (U. S.), there is every reason to believe that these were not cases of true typhus. Large personal observation on the part of the writer, and diligent inquiry amongst the medical officers of the United States army, have satisfied him that as an epidemic, however limited, typhus never prevailed, even at the depots for returned prisoners of war. It is possible that isolated cases of maculated fever may have occasionally happened, but positive proof of such is wanting.

Professor Joseph Jones, in his valuable paper contributed to the Medical Memoirs of the United States Sanitary Commission, recently pub-

* These causes Dr. Parkes arranges as follows :

1. A defective commissariat, especially as to food and fresh vegetables, causing diseases, but mainly predisposing to many more—*e.g.*, malignant malarious fevers, scurvy, and bloody flux. Carthage, 1741; Burmah, 1824; China, 1840.
2. Undertaking military operations in an unhealthy site, and with an unhealthy season impending. Carthage, 1741; San Domingo, 1796; Walcheren, 1747; Java, 1811; American War, 1814; Bulgaria, 1853-54.
3. Exposure to cold, with insufficient clothing and food, giving rise to catarrhs, slight dysentery, rheumatism, and inflammations. Wars of 1742-1760; Crimea, 1854.
4. Propagation of typhus poison, favored by bad ventilation, overcrowding, and filth. Examples as above detailed.
5. Similar propagation of putrid dysentery. Indian campaigns.
6. Propagation of typhoid fever poison and cholera, through the bad sanitary condition of camps, and the occupation of old camping-grounds. Egypt, 1801; Bulgaria, 1853; India.
7. The enlistment of boys as soldiers, whose bones are not yet matured—in place of full-grown men at least twenty-one years of age. Crimea, 1854. (See a short publication *On the Growth of the Recruit and Young Soldier*, by William Aitken, M.D.)
8. Want of cleanliness, excessive use of spirits, and debauchery. [The fact of the great infrequency, or probably immunity from typhus amongst both the United States and Confederate troops during the late civil war, mentioned above, as well as amongst the prisoners in the Southern military prisons, under circumstances which have been generally considered favorable to its production, shows that the etiology of this disease is by no means a simple problem. If crowd-poisoning were the chief factor in its production, all the elements existed at Andersonville and other prisons in perfection. It would seem as if other factors besides the concentrated effluvia from the bodies of healthy persons were requisite for the development of the special poison. Virchow, in a recent lecture on the causes of typhus (*Ueber den Hunger-typhus, und einige verwandte Krankheitsformen*, Berlin, 1868), in which the subject is largely treated, regards the doctrine of its principally arising from overcrowding as altogether too narrow. He contends for the spontaneous origin of the disease, and under circumstances which require closer investigation.]

lished, says: "No case of typhus fever was reported during six months in 42,686 cases of all diseases amongst the Federal prisoners confined at Andersonville, notwithstanding that 40,000 men were crowded upon twenty-seven acres of land, and notwithstanding that all sanitary and hygienic laws were utterly neglected, and the earth was covered with abnormal human excrements and fragments of bread and saturated with urine, and the atmosphere was loaded with stinking effluvia. During the recent civil war I sought for typhus fever amongst the Confederate troops serving in the field, and amongst the general hospitals in various parts of the Confederate States; thousands of sick and wounded were examined with a view to the determination of the existence or non-existence of this disease amongst the Confederate armies; and even the prisoners confined upon Belle Isle, in the Libby Prison, and in Castle Thunder, in Richmond, Va., were not neglected in these examinations, and numerous medical officers of the Confederate army were interrogated upon this subject personally and by letter. No case of true typhus fever came under my observation during the war in any army, in any field hospital, general hospital, or military prison" (p. 600). He further states that the cases entered upon the Confederate sick reports as typhus fever were, in almost every case, if not in all, cases of typhoid fever occurring in those whose blood was scorbutic.]

Phenomena and Symptoms.—Typhus fever attacks persons of both sexes and of all ages, from early infancy to extreme old age, and its advent is somewhat sudden.

After a longer or shorter duration (generally a few days) of unpleasant sensations—in which general soreness, uneasiness, and fatigue without cause, loss of appetite, and disturbed sleep, are the prominent phenomena—the disease begins and advances gradually. It is not possible in all instances to fix the precise time of the commencement of the attack; but in the majority of cases the patient is seized with chilliness, which sometimes amounts to a rigor, usually followed by heat of skin, and occasionally by sweating, pains in the back and limbs, and frontal headache. This headache is a constant symptom, which ceases usually about the tenth day, and always before the fourteenth. During two or three days the chilliness and rigors occur at irregular intervals. The patient alternately hovers over the fire or desires to move from it; and although the skin at the time may be felt hot and burning, he still lingers near the fire-place, and yet again soon complains of the heat of the room; so that he feels when near the fire hot and oppressed, and when away from it chilly and uncomfortable. Loss of appetite, and more or less thirst, exist from the first; the tongue is white, large, and pale, but is afterwards covered with a yellow-brown fur, and is sometimes tremulous, indicating the early loss of muscular power and control. The bowels may be confined or regular; the urine is scanty and high-colored; and nausea with vomiting are often among the earliest symptoms. If sleep is obtained, it is disturbed by dreams, or by the occurrence every few minutes of sudden starts. It is consequently unrefreshing; and although the patient may have appeared to sleep for hours, yet he feels that he has not slept, and declares that he has never closed his eyes. This is the *coma-rigil* of Chomel. On the other hand, there is sometimes a constant ten-

dency to heaviness and drowsiness. The attention cannot be fixed, and the mind ceases to think. A peculiar symptom may now become expressed, to which Dr. Jenner has given more appropriately the name of *coma-vigil* than to that symptom which Chomel has so named. In the *coma-vigil* of Jenner "the patient lies with his eyes open, evidently awake, but indifferent or insensible to all going on around him." This symptom occurred in one-fifth of the fatal cases observed by him. Bodily weakness becomes extreme, and the patient takes to bed by the second or third, and not unfrequently on the first day. While there is absolute loss of muscular power and control, there is at the same time an amount of great exhaustion, disproportionate by its severity to the muscular action. Giddiness and noise in the ears are amongst the earliest and most loudly complained of symptoms. The debility increases rapidly, so that by the seventh day the patient can rarely leave his bed without some assistance. By this time also the want of control over the muscular movements becomes more decided; the legs and arms shake when raised, and the tongue trembles when protruded. The impairment of the mental powers manifests itself in a variety of singular ways. Memory becomes deficient,—the ideas of time are such that it is always supposed to be prolonged. If an event is impressed upon the patient's mind, he will remember it, and it alone. This mazy state of the intellect soon passes into delirium, which becomes manifest first between waking and sleeping, then by night, and finally by day and night. When delirium first sets in, the patient is able to correct himself; if he is made to think, he becomes conscious of his mental error; but his power is soon lost, and delirium becomes predominant.

About the tenth day of the disease, sometimes earlier, the headache ceases simultaneously with the commencement of the delirium; and if it should continue with the delirium, it suggests the probability of some commencing secondary lesion within the cranium, to which special attention must be immediately directed.

The Eruption of Typhus Fever.—About the fifth to the seventh day of the disease the characteristic eruption appears on the skin. It consists of,—(1.) Distinct spots; (2.) A subcuticular rash.

(1.) The *maculæ*, *mulberry*, or *rubeoloid rash*. On the first appearance of this eruption it consists of very slightly elevated spots of a dusky pinkish-red color, somewhat like the stains of mulberry juice. Each spot is flattened on the surface, irregular in outline, with no well-defined margin, and fading insensibly into the hue of the surrounding skin. The spots *disappear* completely on pressure, resuming their distinctive appearances as the pressure of the finger is withdrawn; and they vary in size from a point to three or four lines in diameter. The largest spots appear to be formed by the coalescence of two or more smaller ones; and the shape of the larger spots is more irregular than the smaller ones. After one, two, or three days these spots undergo a marked change. They no longer remain elevated above the surrounding cuticle. Their hue becomes darker and more dingy than at their first appearance. Their margins become more defined, especially on the posterior surface of the body;

and when the finger is firmly pressed on them, they grow paler, but do not entirely disappear. Thus they are said "to fade under pressure;" but they cannot be entirely obliterated, a stain of the cuticle remaining to indicate where they are. A still further change may take place in severe cases. The centres of the spots may become dark purple, unaltered in appearance by the firmest pressure, although their circumferences may fade; or the entire spot may change into a true petechia, becoming of a dusky crimson or purple color, quite unaffected by pressure, with a well-defined margin, and level with the surface. The spots of such an eruption are generally very numerous, close together, and occasionally almost covering the skin. Sometimes, however, they are very few in number, and situated at some distance from each other; and not to be distinguished at first from the *rose spot* eruption. The *mulberry eruption* usually occupies the trunk and extremities, but is occasionally limited to the trunk, and may now and then be observed to extend to the face. After the first, second, or third day after the eruption is apparent, no fresh spots appear, and each spot remains visible from its first eruption till the whole rash vanishes—that is, till the termination of the disease. When very numerous, the eruption, viewed as a whole, has not an equal depth of color. Some places are much paler than others, and the spots have a dull appearance, as if seen through the cuticle. A mottled aspect is thus sometimes given to the skin, on which the darker spots are seated; and hence (2.) A *subcuticular rash* has been also described, which is deepest colored on the most depending parts of the body. From this circumstance the eruption sometimes resembles *measles* so closely as to be distinguished with difficulty from the eruption in that disease. When the spots on the back are of a much deeper hue than those on the anterior surface of the trunk, the skin is at the same time so much congested at the back that slight pressure with the finger leaves a white mark, which slowly returns to its dusky red color. The eruption of the mulberry rash usually appears from the fifth to eighth day of this disease, and subsides between the fourteenth and twenty-first days (DR. JENNER).

Age seems to exert a considerable influence over the eruption; and the following rule has been laid down in relation to this modifying circumstance: In 100 typhus patients under fifteen years of age the rash will be absent in 25. In 100 typhus patients between fifteen and twenty-two years of age the rash will be absent in 14. In 100 typhus patients above twenty-two years of age the rash will be always present.

The spots of typhus fever continue ineffaceably persistent after death.

At the termination of the first, or commencement of the second week, the tongue has a large and swollen appearance, grows dry in the centre, and at the same time its white fur is replaced by pale dirty-brown mucus.

About the ninth or tenth day, or even earlier, the delirium becomes decided, sometimes violent, and always unquiet, although the attention may still be fixed by a sharp question. At this time the patient is in some cases violent, and, unless restrained, leaves his bed to

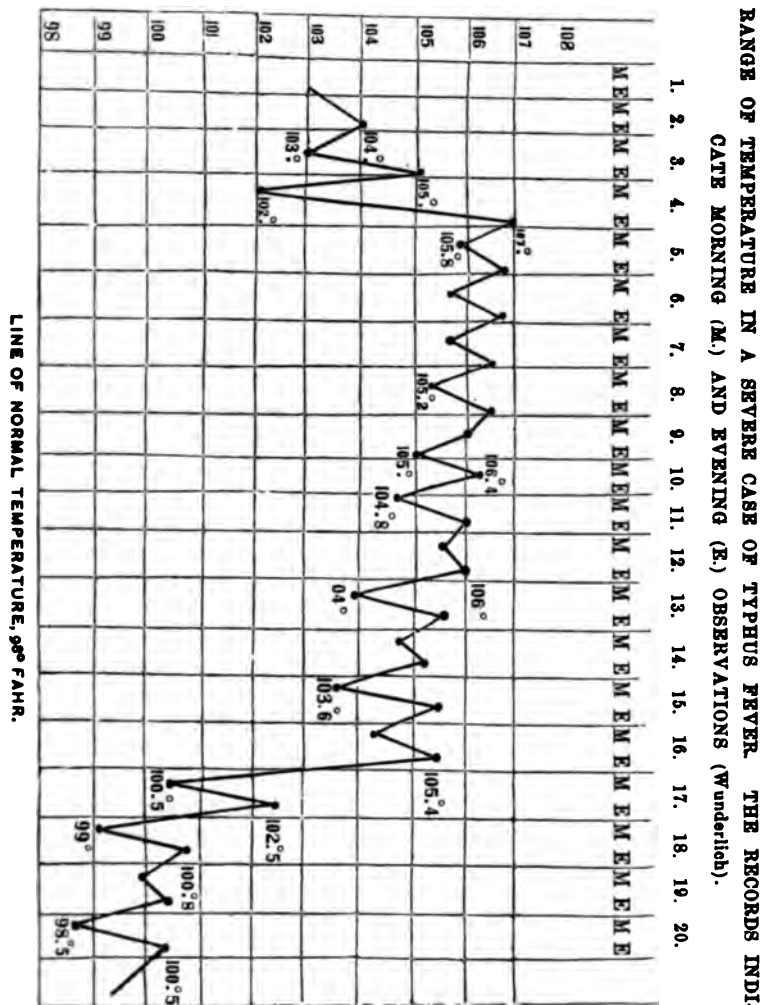
wander about the room. His expression gradually comes to resemble that of a man unwilling to be roused from half-drunken slumbers. It now betokens complete stupidity, oppression, and decided prostration. The complexion, dull and dirty from the first, in the course of the second week becomes absolutely muddy, the conjunctival membranes injected, and the pupils contracted; and the danger of febrile coma, which may supervene, seems very much in proportion to the contraction of the pupil (W. T. GAIRDNER). The face is now often flushed—the flush being dingy and pretty uniform over the whole countenance; but occasionally somewhat more marked on the cheeks than elsewhere.

The eruption gradually becomes darker in hue, the centres of many of the spots, towards the termination of the second week, are unaffected by pressure, and here and there are to be seen some spots with well-defined outline, quite unalterable in appearance by the firmest pressure of the finger. These are true petechiæ. The posterior surface of the trunk is considerably congested, and the spots are there much darker and less affected by pressure than on the anterior surface.

About the tenth or eleventh day somnolence sets in, which may gradually pass into stupor, or even coma, and the expression indicates profound prostration. The patient lies on his back, unable to turn himself in the slightest degree, and the urine is often passed involuntarily, or is retained, requiring the use of the catheter for its withdrawal. The tongue is thickly coated, dry, and dark brown, or even black, appearing as if baked, and perhaps unable to be protruded. The teeth are covered with sordes, the patient is unable to be roused for more than a minute or two, and when so roused he mutters incoherently. The conjunctivæ are intensely injected, and the pupils contracted. The skin is cool and occasionally moist. Miliary vesicles, or sudamina, are sometimes observed about the end of the second week, usually in the groins, at the epigastrium, and under the clavicles. If such vesicles become hard at the summits, then black, and if then the mass drops out as a slough, leaving a circular ulcer, such a vesicular eruption forbodes an unfavorable result (STOKES). The abdomen continues flaccid and indolent throughout. The bowels usually act once or twice a day, the stools being somewhat relaxed.

The pulse, from the outset of the disease, is quickened, and it increases in rapidity in cases which terminate fatally, ranging from 100 to even 150 in a minute; or, after reaching a certain point, its frequency as gradually subsides till health is restored. Cases in which the pulse is remarkably slow are usually cases in which the prostration becomes extreme. In the milder and uncomplicated cases the maximum rate of the pulse is reached before the eighth day, and continues for two or three days at least, at the maximum rate—commencing to decline gradually about the tenth, eleventh, or twelfth day. In the more severe cases the pulse keeps up very high till the thirteenth or fourteenth day, when, if the disease is about to end favorably, there is a *sudden* and marked fall (say from 120 to 96, or from 100 to 84), indicating a decided crisis (PERRY).

The skin throughout the whole course of typhus fever is often particularly sensitive, the slightest touch occasioning pain. The heat of the skin conveys also a burning pungent sensation—the temperature ranging from 102° to 107° Fahr. Greisinger, of Zurich, after insisting on the non-identity of typhus and typhoid fevers, has described the ranges of temperature in cases of typhus as particularly diagnostic and characteristic (*Arch. der Heilk.*, vol. ii, p. 557, 1861). Dr. Cheyne, of Dublin, recorded 109° Fahr. as the highest he observed, and a few days before death he observed the temperature to fall to 95° Fahr.



The course of typhus fever, although it may have some features in common with that of enteric or typhoid, yet shows great and numerous differences. In typhus, the fever, as denoted by the tem-

perature, is maintained continuously for a longer time without interruptions.

If the temperature at the commencement before the fourth day does not exceed on any evening 103.5° Fahr., the fever may be expected to run a mild course; and more especially if the increase of temperature takes place moderately, and is of limited daily duration during the beginning of the second week. In *mild* cases a small abatement of temperature takes place on the fourth day, which becomes more obvious by the seventh day; and this abatement is followed, at the commencement of the second week, by a small re-increase of very limited duration, after which the tendency to further decrease reappears, and by the end of the second week, or the beginning of the third, the normal temperature is again attained. On the other hand, in *severe* cases the fever continues with great intensity, at least to the twelfth day, and mostly throughout the whole of the second week.

Defervescence rarely takes place before the first half of the third week. In very severe cases the temperature may rise above 104.7° Fahr., and it frequently reaches 106° Fahr. or more. The maximum of temperature is usually attained *before* the ninth day (GRIMSHAW), or *on* the ninth day (WARTER); and according to Grimshaw the extreme height of the thermometer in typhus seldom exceeds 104° , occasionally reaches 104.5° , and may, in exceptional cases, rise to 105° —a conclusion nearly coinciding with the observations of Dr. Perry, of Glasgow. The differences between the morning and the evening temperatures amount, during the fastigium (from the middle of the first to the middle of the second week), rarely to more than 1° Fahr.; and from the middle of the second week to a difference of about 1.5° Fahr. Greater differences happen only temporarily—a character which distinguishes typhus from enteric or typhoid fever, so far as ranges of temperature are concerned. But the difference between typhus fever and enteric or typhoid fever is rendered still more striking by their respective modes of defervescence. In typhus fever the defervescence shows no gradual remissions, as in enteric or typhoid fever; on the contrary, the defervescence of typhus fever is invariably sudden. When the fall is great (3° to 5°) as well as sudden, critical diarrhœa may be severe and serious, as in some of the cases detailed by Dr. Grimshaw; and a sudden fall of temperature to a range below the normal, other symptoms being serious, is a bad sign, and may forebode death. It takes place precipitantly, and the normal temperature is reached sometimes in one night, mostly in twenty-four or thirty-six hours.

The disease generally terminates, if it proves fatal, from the twelfth to the twentieth day; and before death the prostration increases to the last degree. The average duration of Jenner's fatal cases was fourteen days; of Reid's, thirteen days. *Subsultus tendinum*, or involuntary twitchings of the muscles of the face and arms, make their appearance. The face becomes dusky or even livid, and the breathing very quick, the pulse becoming so rapid and feeble that it can scarcely be felt. Some want of resonance of the most depending part of the chest may often be observed at this

stage of the disease. The respiratory murmur at the same part becomes muffled, as if heard through a covering, and there is sometimes a little coarse, unequal crepitation. The urine, which is *now* secreted in large quantities—from three to four pints daily—is restrained, or passed into bed with the stools involuntarily. The skin is bathed in a profuse sweat, and the temperature is apt to fall below the natural standard. The patient lies on his back unable to move, or he sinks towards the bottom of the bed if his head be in the least elevated. Towards the middle or end of the second week a slough may form on the lower end of the spinal region, or on the region over the posterior spine of the ilium.

For a day or two before the fatal termination, the condition termed *coma-vigil* may come on. In this condition the patient never sleeps. He lies on his back with his eyelids widely separated, his eyes staring and fixed in vacuity, his mouth partially open, his face pale and expressionless. He is totally incapable of being roused to give a sign of consciousness, the breathing is often scarcely perceptible, the pulse rapid and feeble, or unable to be felt, the skin cool, perhaps bathed in perspiration. Life is only known to have ceased by the eye losing its little lustre, and the chest ceasing to effect its slow and feeble movements. Dr. Jenner has never seen recovery from this condition. Death generally takes place without any return to consciousness, and by syncope rather than coma (MURCHISON).

If the disease should terminate in recovery, the improvement in the condition of the patient is frequently sudden. Some time between the thirteenth and the seventeenth day he may fall into a profound quiet sleep, lasting for several hours; and generally after from twelve to twenty-four, or even more hours, he awakes decidedly improved in all respects—indeed, quite another man. At first he is bewildered or confused, and wonders where he is; but he may recognize his attendants and friends, and is conscious for the first time of his extreme debility. The complexion is clearer, the delirium has disappeared, the pulse has fallen in frequency and gained in strength, the conjunctivæ are no longer injected, the tongue is moist at the edges; there is perhaps a little appetite, the skin is softer and moist, and the spots paler. His limbs retain their sensibility, but when he attempts to move them, they seem at first as if separated from the body, so great is the prostration induced by typhus fever (MURCHISON). In a few days the tongue cleans completely, the appetite becomes ravenous and insatiable, and the patient rapidly regains strength. Dr. Jenner considers the duration of the disease to be measured by the duration of the eruption; and the average duration of cases that recover, he states to be from fourteen to twenty-one days; although, not unfrequently, in very mild cases, the fever terminates before the fourteenth day. After twenty-one days local lesions sufficient to cause death were always discovered in fatal cases of *typhus*. In other words, after the twenty-first day, death does not then occur *from the fever alone*, as may be the case before the twenty-first day. There are two very opposite circumstances under the influence of which the date of the first appearance of the

eruption is changed, and its duration shortened. These are,—(1.) A very mild attack of the specific disease; (2.) The development of severe local complications in the course of the specific disease. There are cases of *typhus fever* which appear to die within a limited period after the outset of the illness, from the direct action of the poison on the blood or nervous system, and with the entire absence of local lesion, so that even the skin is not affected with eruption.

Condition of the Blood.—In *typhus fever* the microscopical characters of the blood are often such as to prove a marked deviation from its normal state. Amorphous heaps of red discs replace the normal rouleaus, and the adhesion of the red discs to each other, in the imperfectly formed rouleaus, is far less complete and long continued than in healthy blood. The red discs part with their coloring matter more easily and dissolve more rapidly than they do in their normal state. This is shown by the red serosity found in almost every serous cavity, the deep dusky-red hue of the flesh, and of every structure in contact with the blood. The blood drawn during life, or found after death in the vessels, is loosely coagulated or absolutely fluid (DR. JENNER). It is also more apt to become putrid, when taken from the body during life, than healthy blood, or than blood in other diseases. According to Lehmann, the salts are increased rather than diminished; and there is good reason for believing that the unnaturally fluid state in typhus fever results from an abnormal amount of ammonia, possibly derived, as Dr. Murchison suggests, from the decomposition of urea; and there is evidence, as Drs. Richardson and Murchison have shown, that the blood of typhus fever contains an increased amount of ammonia.

Secondary Lesions and Complications of Typhus Fever.

1. Convulsions and Cerebral Affections.—The most formidable, and fortunately the most rare, is the occurrence of convulsions. When they do occur, the case almost invariably proves fatal, unless the convulsions occur in a patient who has suffered from epilepsy; and the subjects of them seldom present any morbid appearances after death sufficient to account for their occurrence. In *The Edinburgh Monthly Journal* for June, 1848, the details of six cases of typhus fever are given, five of which proved fatal by convulsions, in different wards of the general hospital there, and all of them within a period of twenty-one days, in the months of January and February. The following are the days of the fever at which convulsions are stated to have occurred, the number of hours they continued to recur, and the apparent mode of death:

No. of Case.	Day of Fever.	Number of Hours the Convulsion Recurred.	Mode of Death.
1st. <i>Edin. Mon. Journal</i> ,	7th	2	Coma.
2d. " "	11th	5	Coma.
3d. " "	15th	6	Coma.
4th. " "	14th	24	Coma.
11th. Of Mary G—, related by Dr. Jenner,	11th to 13th	72	Coma.
13th. Of Thomas B—, related by Dr. Jenner,	9th	36	Coma.

The occurrence of convulsions in such cases may be fairly referable, in the present state of our knowledge, to the morbid condition of the blood in *typhus* fever, and the altered condition of the nervous system which ensues; and probably they have always a uræmic origin. With reference to the absence of any appreciable lesion in the brain, as in these cases, it may be remarked that our usual instruments of research, applied to the nervous texture, are insufficient in all instances to indicate disease, even where it does undoubtedly exist. There are, for instance, physical conditions of texture, which are of the utmost importance in pathology, such as the specific gravity, and which are appreciable by the proper means and instruments of research, even when the tissue of the organ presents to our senses of sight and touch no external evidence of disease.

The cerebral complications are generally attended with what are commonly called "head symptoms," and are preceded by long-continued high temperatures. Dr. Jenner very emphatically calls attention to the fact, that the continuance of the headache complained of spontaneously after the commencement of delirium is generally indicative of increased vascular action within the cranium. It may also be noticed that the headache which precedes the delirium is often in such cases of a very severe and constant kind, the face being sometimes pale and sometimes red, and greatly expressive of the distress the patient suffers. The eye, haggard or brilliant, with its conjunctiva injected and its pupil contracted, is painfully sensible to the light, and is therefore generally closed. The least noise is insupportable, and the patient is troubled with noise in his ears. His temper is altered, and his answers short and fretful. This condition is that of increased excitement, but not as yet of delirium, and, supposing the membranes of the brain to be inflamed, denotes diffuse inflammation of those tissues. At the end of a period of time, varying from two to ten days, the patient becomes delirious. His delirium may assume every character,—joyous or melancholy, furious or tranquil; and in some cases he wanders from subject to subject, while in others he incessantly recurs to the same theme, and even to the same few words. In others, though the cases are few, the disease assumes every character of insanity; and, if permitted, the patient, confined in a strait waistcoat, presents the extraordinary spectacle of being able, in typhus fever, to walk about the wards. The phenomena of this stage show that the inflammation of the membranes of the brain has extended to the substance of the brain itself. The commencement of effusion is indicated by the active delirium changing into a low muttering (*typhomania*), by the patient no longer requiring restraint, by his muscles becoming spasmodically affected with slight twitchings, or *subsultus tendinum*, showing how rapidly the nervous power is exhausted, and how feebly supplied; also by the pupil of the eye becoming expanded or contracted; by the feces being passed involuntarily; by the urine being retained; and by the rapid grouping of those other symptoms so happily described by Shakspeare as "the stony coldness of the feet creeping upward and upward," "the babble of green fields," and

the "fumbling of the bed-clothes," all indicative of approaching death. Intra-cranial serosity is generally decidedly increased; and hemorrhage into the arachnoid occurred in one-eighth of Jenner's fatal cases. When the patient recovers, however, from this stage of cerebral complications, the appetite improves, the pulse becomes fuller and steadier, the countenance more tranquil, the mind firmer, and the sleep natural, till at last convalescence is fully established.

The respiratory movements are often influenced by this cerebral condition. In the first week they do not exceed twenty or twenty-four in the minute; but when delirium supervenes and the pulse increases in frequency, they often rise to thirty or more, without any pulmonary lesion. In cases of great cerebral disturbance the respirations become sighing, irregular, spasmodic, or jerking, and then coma is apt to supervene. The "nervous respiration" of Dr. Corrigan, or what is sometimes also called "cerebral respiration" is denoted by a blowing or hissing sound in breathing, the lips being kept closed; the cheeks are distended, the nostrils dilate with each expiration, and the breath is forced through the closed lips with a puffing, blowing noise. Such breathing is irregular, a long pause being followed by a deep inspiration, and subsequently by short respirations in rapid succession. In some cases the action is entirely diaphragmatic, the respiratory muscles of the trunk being paralyzed (MURCHISON). The air expired has a disagreeable odor, most marked in the advanced stage of severe cases. It resembles the *typhus odor* exhaled by the skin, and which has been compared to the "odor of rotten straw," to the "smell of mice, deer, and certain reptiles," or to the smell of "the leaves of rue when rubbed between the fingers." By some it is spoken of as "pungent, ammoniacal, and offensive;" but is not to be confounded with the smell from urine passed in bed. It seems to be, however, a smell *sui generis*, as Murchison very justly observes; and nurses experienced in typhus fever are quite familiar with it, and are able to distinguish cases of typhus fever by this peculiar typhus odor alone, which is always strongest in damp weather, and when the ventilation is bad; and it is highly probable that the typhus poison is contained in this odoriferous substance. The expired air of typhus patients contains a smaller quantity of carbonic acid and a larger amount of ammonia (MURCHISON, pp. 134-137).

2. Secondary Pulmonic Complications are not uncommon in *typhus fever*, in the form of *pneumonia*. The congestion of the blood, in the posterior parts of the lungs, may give a tendency to this; and its presence may be suspected from the livid expression of the face, the existence of cough with rusty expectoration, the diagnosis being confirmed by the usual auscultatory means. It is seldom that pain is complained of. The part of the lungs affected for the most part is that which rests against the hollow of the fourth, fifth, and sixth ribs, between their tubercles and angles; the position of the patient appearing to determine the place of consolidation. Such consolidations are not to be regarded as analogous to what we see in an ordinary pneumonia, occurring in an otherwise healthy person. The incubation of the lesion is latent, and the symptoms are masked,

and the nature of the complication is only to be recognized by careful physical examination and determination of temperature, which suddenly rises and continues high. The full expression of the morbid state is often for some time undecided; the exudation, being of a serous nature, is slow to solidify on the one hand, and yet the symptoms of resolution do not appear on the other. Dr. Hudson, of Dublin, attaches some importance to a certain tympanitic resonance, which becomes manifest over the diseased lung, as a sign of the existence of the pulmonic lesion. He describes it as "a tympanitic clearness over the solidified lung, without air being present in the pleura." Dr. Lyons explains this abnormal clearness as the result of the increased pressure of the respiratory column of air in the permeable portions of the pulmonary lobules, which become expanded beyond their natural volume, and thus a condition of temporary emphysema is produced, which yields a clear sound on percussion (Dr. Stokes, in *Medical Times and Gazette*, May 26, 1855).

In some cases of pulmonary lesions there appears to be a combination of circumstances which leads to a fluid or purulent state of the diseased part, resembling the third stage of pneumonia as described by Laennec. The conditions which lead to this form may be stated to be,—(1.) A sudden exudation and abundance of fluid matter; (2.) A great amount of tissue involved; (3.) Diminished vascularity and consequent (4.) Abeyance of absorption, tending to (5.) Increased fluidity of the diseased part; (6.) Breaking up or solution of the young and growing elements. A lung in this condition seems to have passed, as it were, at once into this state, without any well-marked hepatization.

3. Gangrene of the Pulmonary Tissue is by far the most formidable of the thoracic secondary lesions of *typhus fever*. The hepatization of the lung is not, as in the last instance, obscure, but the consolidation is at once sudden, complete, and extensive, involving perhaps the greater part of the lung, and coming on without any marked physical signs different from what are to be heard in the simple congestion of *typhus*. A gangrenous cavity forms in the substance of the solidified mass, and is only indicated by the fetid expectoration and the accompanying physical signs of a cavity. Large eschars are apt to form towards the pleural surface, surrounded with well-defined lines of demarcation where separation of the slough proceeds. In this gangrenous slough every simple element of the pulmonary tissue becomes disintegrated, almost perfectly liquefied; and sometimes it happens that the gangrenous cavity does not communicate with the bronchial tubes, and then the morbid state is difficult to diagnose, and its existence is often unknown till after death. With physical signs, the expression of the countenance of the patient is often highly suggestive. It suddenly becomes small, pinched, contracted, ghastly, miserable and death-like. The eyes are sunk and void of lustre; and, along with languor, the patient feels nausea, and sometimes vomits. There may be several distinct gangrenous centres, as if the lesion had been, from the first, disseminated or lobular.

4. Secondary Cardiac Lesion.—This lesion assumes the form which

Dr. Stokes has called "typhus softening of the heart." He is inclined to consider that the muscles of the larynx and the circular muscles of the trachea are sometimes similarly affected, as well as the involuntary muscles generally. This complication has for the most part occurred when there was a great amount of the secondary bronchial disease. The wasting of the involuntary muscles is always great in typhus. In the heart it is more obvious than in the arterial or systemic portion.

The cardiac phenomena of typhus (adynamic) are chiefly indicated by a diminution of the impulse, and an impairment or loss of the first sound—the impulse diminishing progressively from the fifth or sixth day to the termination of the disease—while the systolic sound becomes daily more feeble or quite inaudible, leaving the second sound clear and distinct.

The poison, however, does not necessarily make itself manifest through all the series of local secondary affections already referred to. Thus, in one year the lungs will be attacked in every case; in others, the bronchial membranes or the membranes of the brain; while in other years such attacks will be rare,—the exception, and not the rule of the disease.

Prognosis.—The occurrence of certain phenomena, or the presence of certain symptoms, indicating the existence of complications, are the main guides to an opinion. These may be arranged in the following summary under three heads, namely:

1. Combinations of Symptoms and Phenomena which are of extremely unfavorable import.—(1.) A presentiment of death on the part of the patient; (2.) A pulse of 120, which at the same time is soft and compressible, small, fluttering, irregular, intermittent, reduplicate, or imperceptible; (3.) Complete absence of cardiac impulse, and an audible systolic sound; (4.) An excited or thumping action of the heart, associated with a very feeble radial pulse; (5.) Hurried respiration, whether "cerebral" or due to pulmonary lesion; (6.) Sleeplessness, associated with delirium, both of which are persistent; (7.) Severity of cerebral symptoms, and these symptoms coming on early; (8.) The occurrence of complete "coma-vigil;" (9.) Extreme contraction of the pupil; (10.) Extreme prostration occurring early; (11.) Muscular tremor, picking and catching at the bed-clothes, subsultus, and spasmodic twitchings of the muscles of the face; (12.) Urgent and protracted hiccup; (13.) Rigidity of the muscles of the limbs, and squinting; (14.) Relaxation of the sphincters before the tenth day; (15.) Retention of urine; (16.) Tympanitis, with extreme nervous prostration; (17.) A dry, brown, hard, retracted, tremulous tongue; (18.) The more abundant and darker the eruption the greater the danger and severity of the case; (19.) Great lividity of the face and extremities, and a dusky erythematous condition of the skin on the dependent parts of the body; (20.) Continuous profuse sweating, coldness of the surface, cold breath, and a rapid weak pulse; (21.) A sudden diminution in the amount of the excretion of urea; (22.) The occurrence of blood or albumen in the urine before the tenth day, especially when associated with casts of the uriniferous tubes; (23.) Pulmonary hypostasis and bronchitis, pneumonia,

gangrene of the lungs, convulsions, pyæmia, erysipelas, parotid swellings, inflammatory swellings, bed-sores, gangrene, renal disease, scurvy, the gouty diathesis.

2. Combinations of Symptoms or Phenomena which may be regarded as of favorable import.—(1.) A sudden fall in the frequency of the pulse; (2.) When a patient, after lying for days on his back, helpless and motionless, manages to turn himself round and sleep on his side, or if he is able to draw up his leg and rest it on the foot in the flexed position in the bed; (3.) Cases without rash, or in which the rash is scanty; (4.) When the excretion and elimination of urea and uric acid continue free and copious; (5.) Sudden cessation at the end of the second week of several of the unfavorable symptoms and phenomena; (6.) Diminution of the rapidity and increase in the strength of the pulse; (7.) A slight return of appetite, while the tongue becomes clean and moist at the edges; (8.) A diminution of the dusky tinge of the face, a less stupid appearance of the countenance, and a less injected state of the conjunctivæ, with signs of returning intelligence.

3. Modes of Fatal Termination.—(1.) Death during the primary fever may occur from syncope or from coma. In the former case the heart's action is enfeebled from paralysis or disease of its muscular tissue. In the mode of death by coma the blood has undergone such modifications as render it incapable of supporting the changes essential to existence. Its contamination seems mainly due to the admixture of urea and other products of the retrograde metamorphoses of tissue, and from the diminution and destruction or solution of its red corpuscles. (2.) Death is for the most part due to a combination of syncope and coma; and, as a rule, the patient is quite unconscious for a considerable time prior to death. (3.) Death may occur from one of the many complications which happen before or after the cessation of the primary fever.

Morbid Anatomy.—The morbid anatomy of cases of typhus fever has been carefully investigated by Gerhard and Pennock, A. P. Steward, John Reid, Thomas Peacock, William Jenner, Felix Jacquot, Barrallier, and Murchison. All are agreed that there is no constant nor characteristic lesion; and they may be summed up generally as follows: "A fluid condition of the blood; hyperæmia of the cerebral membranes and increase of intra-cranial fluid; bronchial catarrh and pulmonary hypostasis; softening of the heart, liver, spleen, and pancreas; hyperæmia and hypertrophy of the kidneys" (MURCHISON, p. 245).

Treatment of Typhus Fever.—Before considering the treatment of typhus fever, it is of the greatest importance to be aware of the changes which go on in the system during its progress. Dr. Parkes has observed the nature of these changes in a most conclusive manner. His observations are of great scientific interest, and of important practical bearing ("Gulstonian Lectures," in *Medical Times and Gazette* for February 28, 1857). In an uncomplicated case of typhus fever the body loses flesh rapidly, owing not only to diminished ingress of food, but also to increased egress of bodily structures in the form of excretory products. The metamorphosis of tissue,

as judged by the urine, is augmented. The only complete analysis of the urine in an uncomplicated and undoubted case of *typhus fever*, when no medicine whatever was given, is an analysis made and recorded by Dr. Parkes (*Urine in Disease*, p. 258). "The condition of the urine," he writes, "was that of ordinary pyrexia. The water was lessened; the urea was increased one-fifth; the uric acid was in large amount, and spontaneously, or on the addition of an acid, deposited. The chlorides were entirely absent; there was no diarrhoea or sweating; the sulphuric acid was rather high; the phosphoric acid was not determined. The free acidity was very slight; and (differing from many pyrexiae) the pigment and extractive matters were throughout in small amount. The urea continued large, and the chloride of sodium small in amount, for some days after the temperature had fallen to below the normal limit. The excretion of urea was remarkably regular in amount from day to day; for during ten febrile days its range was only 15 grains (1 gramme) below the mean of the ten days, and 20 grains ($1\frac{1}{2}$ gramme) above it. And this took place with great alterations of temperature. It then, as usual, fell during convalescence, and rose again to the healthy standard in three or four days. The chloride of sodium was clearly retained in this case, for there was constipation, and the skin was dry, so that none could have passed off by the intestines or surface." "It would seem also," he further observes, "that the urine in typhus is much more frequently albuminous than in typhoid fever."

In three cases of typhus fever associated with jaundice (which is extremely rare in typhus cases), Dr. Murchison examined the urine, which was also jaundiced. There was no reaction on testing for the bile acids; but in two of the cases *tyrosin* and *leucin* were found. In one of these cases the urine was almost devoid of urea. At the autopsies of two of them there was no derangement of the biliary ducts (*Path. Society*, Feb. 3, 1863).

The following inferences are drawn from the table given by Dr. Parkes:

1. In spite of the many pints of fluid drank, a small quantity of water left the system by the kidneys and skin, and none at all by the bowels. This retention of water is not peculiar to typhus, and its cause is quite unknown.

2. The amount of urea was greatly increased. The normal amount of urea excreted by active men on good diet, between twenty and forty years of age, weighing 145 lbs., is 491 grains in twenty-four hours. A boy ill of typhus, aged seventeen, weighing not more than 129 lbs., excreted not less than 532 grains daily, although he was on fever diet, and taking scarcely any nitrogenous food.

3. The chloride of sodium is excreted in health at the rate of 180 grains daily. In this case of typhus fever it was present only in traces, the amount being too small to be determined. Like the water, this retention is common to the pyrexiae.

4. Metamorphosis of tissue was more active by one-fourth daily.

General Indications for Treatment.—From most careful observations such as these, Dr. Parkes thus gives an outline of the principles upon which fevers are to be treated. The treatment of fever (and

typhus and *typhoid* fevers are not exceptions) may be summed up as being a combination of measures to *reduce excessive heat*, to insure *proper excretion*, and to *act on the semi-paralyzed nerves*; and, as Dr. Murchison justly observes, "every remedial agent which shall be found to promote the elimination of urea, without increasing the destructive metamorphosis of tissue, will deserve a trial in typhus" (*l. c.*, p. 268).

To reduce heat and to regulate elimination are but secondary indications in the treatment of *typhus* fever, compared with the influence which must be exercised over the nervous system; and one of the greatest objects of therapeutics at the present day is to find substances which will act on the nerves and the blood, and restore them in some way to their normal action. (See page 153, *ante*.)

Special Indications for Treatment.—Our objects in the treatment of typhus fever should be,—(1.) To neutralize the poison and to correct the morbid state of the blood; (2.) To eliminate the poison and the products of the destructive metamorphosis of tissue; (3.) To reduce the temperature; (4.) To sustain the vital powers, and to obviate the tendency to death; (5.) To relieve the distressing symptoms; and (6.) To avert and subdue local complications (MURCHISON, p. 265).

1. In the belief that the morbid condition of the blood in typhus fever may be due to the presence of ammonia in some as yet unknown combination, the use of mineral acids has been recommended by many physicians. Murchison considers their beneficial effects in typhus as undoubted, and in this opinion he is confirmed by the experience of Huss of Stockholm, Haller of Vienna, and of Mackenzie, Chambers, and Richardson, in this country. Huss recommended phosphoric acid in doses of ten to fifteen drops every second hour, believing that the phosphorus exerts a special influence on the brain; but in the advanced stage, and especially if sweating, numerous petechiæ, or ecchymoses be present, he has recourse to sulphuric acid in doses of fifteen to twenty drops every hour or every second hour. Hydrochloric acid is preferred by Drs. Murchison, Richardson, Mackenzie, and Chambers. It may be given to the extent of one fluid ounce of the dilute acid, mixed in a quart of barley-water, sweetened with syrup of ginger, and flavored with lemon-peel. Dr. A. P. Stewart has used with advantage the *tinctura perchloridi ferri*, in doses of thirty minims every three hours. Dr. Murchison recommends *nitromuriatic acid*. He prescribes twenty minims of *hydrochloric acid* with ten minims of *nitric acid* every three hours, each dose being diluted with the patient's drink. But if the "typhoid state" is developed in a marked manner, *dilute sulphuric acid*, in doses of fifteen to twenty minims every three hours, in combination with *ether*, and small doses of *quinine*, are to be had recourse to as in either of the following formulæ:

R. Acid. Hydrochlor. dil., ℥xx; Acid. Nit. dil., ℥x; Spt. Æther. Nit., ℥lx; Liquor. Cinchonæ, ℥xxx; Decoc. Scopar. comp., ʒj; *misce*. A draught so composed may be administered every third hour.

Or, R. Quinæ Sulph., gr. ʒ; Acid. Sulph. dil., ℥xx ad ℥xxx; Æther. Sulph., ℥xv ad ℥xxx; Syrup. Aurant., ℥lx; Decoc. Scopar. comp.,

℥j; *misce*. A draught so composed may be administered every third or fourth hour.

When the acids are cautiously administered in smaller doses, in conjunction with a few minims of solution of muriate of morphia, if the bowels be irritable, sweetened with syrup of orange-peel and diluted with water, the draught is generally relished, and the tongue, from being dry, and hard, and brown, becomes moist and clean (PERRY).

2. To insure proper excretion and elimination in fever is much more difficult than to reduce temperature, which, for obvious reasons, it is not always wise to attempt the reduction.

Perhaps the best general method to insure proper excretion is to supply the system with abundance of *alkaline salts*, which are not now given in the food, and to maintain the action of the kidneys, the bowels, and the skin. *Chloride of sodium*, the *alkaline salts of potash*, and probably also those of *soda*, tend to aid the formation of urea and its elimination. In the use of *nitrate of potash* and of *iodide of potassium*, which are not natural constituents of the frame, Dr. Parkes has observed that, at the first employment of these, there is often a marked lessening of excretion, as if the chemical processes then going on in the body had been interfered with, for afterwards the elimination again increases, as if the system had accommodated itself to the remedy.

Purgatives tend to insure a proper excretion, probably by removing from the blood some of the abnormal products formed in fever. The great relief which sometimes follows their use, as well as the fall of temperature, seems to show this. Where there is retention of urea, they aid its elimination, because we know that urea passes off sometimes by the mucous membrane of the stomach and bowels. The patient should be allowed to drink freely of water; and five grains of the *nitrate of potash* may be given with each dose of the nitro-muriatic acid already mentioned. Dr. Murchison recommends *nitre whey*, prepared by boiling ℥ij of nitre in a pint of milk, and straining; or a drink prepared by dissolving ℥j to ℥ij of the *bitartrate of potash* in a pint of boiling water, and flavored with lemon-peel and sugar; but if the patient be very prostrate, or if the bowels be relaxed, *nitric ether* is to be substituted for the *nitrate of potash*.

Tea and coffee have been recommended in the stupor of typhus; and it is probable, as Dr. Parkes has shown, that their good effects are due to their power of eliminating the urea already formed in the blood. The coffee may be given as an extract, or as a strong infusion of the powdered berry made in the ordinary way. Tea has been recommended, as an infusion of the green tea leaf. As beverages or common drinks in fever, both tea and coffee have been found to relieve the headache, the pulse becoming fuller and stronger under their use. Böcker, L. Lehmann, and Hammond, all agree in showing that in health they greatly lessen the urea (Parkes *On the Urine*, p. 76). With respect to *chloride of sodium*,

Dr. Murchison recommends that large quantities of this salt should be given with the beef tea.

The action of the bowels is to be maintained by emetics and laxatives. In the first instance, if the patient is seen early—*i. e.*, before the sixth day—an emetic of ipecacuanha (one scruple), and of antimony (one grain), or of carbonate of ammonia (two scruples), in place of the antimony, is to be administered. If the bowels remain confined after the emetic, a mild laxative of rhubarb and calomel, or of castor oil, is to be given; and failing these, or in place of them, a simple enema is to be administered (MURCHISON, p. 269). The advantages of emetics are, that they relieve the patient to some extent by mitigating or removing headache and general pains. They also reduce the temperature, abate thirst, and quiet gastric disturbance.

Emetics, however, are contraindicated if the patients are unusually weak, or if the disease has advanced beyond the first week. Laxatives and enemata, however, ought to be repeated daily, if required, so as to secure a motion of the bowels once a day. In this respect the treatment is different from the treatment which ought to obtain in typhoid fever, as already mentioned. Excrementitious matters in the intestines must be removed by gentle aperients. The dark offensive matters accumulated in the intestinal canal in typhus fever may have a secondary deleterious effect on the system if they are allowed to remain. Purging, however, is to be avoided, and *fresh-made* compound rhubarb pill mass, which *tends to stimulate the peristaltic action of the intestines*, is as good a medicine as can be given, followed, if necessary, or alternated, by a small dose of castor oil, or by a simple enema.

Diaphoresis is not to be encouraged beyond the insensible transpiration of the skin; and to remove which the wholesome detergent of tepid water sponging is most beneficial. It ought to be used twice or three times daily, and quantities of *Condy's fluid* or of *muratic acid* (3j ad Oj) may be mixed with the tepid water (MURCHISON). The measure is a good one in a hygienic point of view, and it contributes—

3. To reduce temperature, for which the external application of cold water was once practised to an extreme degree by Currie, as originally recommended by Dr. Robert Jackson. In health such an application as that of cold water has a great effect in reducing temperature, and tends to increase metamorphosis (LEHMANN, SANDERSON).

4. The vital powers are to be sustained by food in the first instance. For this purpose, nourishment ought to be given often, and at stated intervals—at least once every three or four hours after the fourth day of the fever. Even if the patient is asleep, or seems to be so, he must be roused at these stated intervals (not oftener) to take his food or his stimulants. But if, towards the period of the crisis, the patient appears to be in a sound sleep, he ought not to be disturbed. The indications for treatment just described apply to the earlier stages of the fever, up till about the fourteenth day.

Alcohol in small quantities, as well as tea, coffee, and other substances, have a directly stimulant action on the nervous system, and on the organs of circulation; at the same time, they diminish the metamorphosis of the tissue-elements. Few remedies, however, require more discrimination in their use; and the following guides for their administration are compiled from the careful observations of Dr. Murchison (*l. c.*, p. 269):

1. Wine is not usually required during the first five or six days of the illness, but most cases require some stimulants during the second week; and, as a rule, the physician may find it necessary to begin to administer stimulants about the seventh or eighth day.

2. The indications for the administration of alcoholic stimulants are mainly derived from the state of the organs of circulation; and the profession is indebted to Dr. Stokes (1839) for pointing out the importance of cardiac and radial pulses as guides for the use of alcohol in fever.

These indications are,—

(a.) Extreme softness or extreme hardness and compressibility of the pulse. An irregular, intermitting, or imperceptible pulse more imperatively demands stimulants than a merely rapid pulse. So also an abnormally slow pulse—*e. g.*, 40 to 60—is a stronger indication for stimulation than a quick pulse.

(b.) When the cardiac impulse becomes weak, and when the first sound is impaired or absent, a liberal allowance of stimulants is demanded: and in every case where there are doubts as to the propriety of giving stimulants, the heart must be examined with the hand and with the stethoscope, because the state of the pulse alone is not sufficient to judge from. The impulse may be found to diminish progressively from the fifth or sixth day to the termination of the disease; and for several days prior to death or recovery it may be entirely absent. The systolic sound of the heart becomes daily more feeble, and ultimately may be quite inaudible, leaving the second sound clear and distinct; and before the first sound is altogether lost, it may become so short that it is difficult to distinguish it from the second sound. If the action of the heart be rapid, its sounds may thus come to resemble closely those of the *fetus in utero*. A violently excited heart all through the disease, with cold surface, cold breath, and feeble pulse, demands wine from the first; but even with its judicious use the prognosis in such cases is extremely doubtful (STOKES, GRAVES, MURCHISON).

Other indications for stimulants may be stated as follows:

(c.) If by raising the patient to his semi-erect position a tendency to syncope is induced, or great prostration is manifest, with diminished strength and volume of the pulse, then stimulation must be commenced.

(d.) The darker and more copious the eruption, the more is the necessity for stimulants, especially if petechiae are numerous.

(e.) Profuse perspiration, with no improvement in the general symptoms, requires an increased supply of stimulants.

(f.) Coldness of the extremities, stupor, low delirium, tremor, subsultus, involuntary evacuations—symptoms generally of the

"typhoid state"—are indications for the liberal administration of alcohol; but the propriety of giving stimulants in delirium depends on the state of the pulse. If, on the trial of stimulants, the patient becomes tranquil, they do good, and may be continued; if the reverse, their use must be suspended.

(g.) A dry brown tongue is an indication for wine or brandy, and if it becomes clean and moist at the edges under the use of either, such stimulation is beneficial.

(h.) Complications, as a rule, increase the necessity for stimulation; and large quantities of stimulants are called for if *pyæmia*, *erysipelas*, *bronchitis*, *pulmonary hypostasis*, *pneumonia*, *inflammatory swellings*, *bed-sores*, or *local gangrene* should supervene.

(i.) Persons who have led intemperate lives, and old persons, require stimulants early in the fever, and in large quantities.

The effects of alcoholic stimulation require to be most carefully watched throughout the whole period of their administration. Four ounces of wine in the twenty-four hours is enough to begin with; for if the blood be overloaded with the products of alcoholic ingestion, further alcoholic stimulation will lead to increased contamination, and it is rare that more than eight ounces of brandy in twenty-four hours are necessary.

There are differences in the demand for stimuli in the typhus of different countries, and in the fever of different epidemics. Dr. Wood tells us that in America cases requiring wine or brandy are extremely rare. Dr. Stokes says that the *typhus* in Ireland demands large quantities of wine. In Scotland, also, wine is the great mainstay in the treatment of *typhus fever*, requiring often to be administered largely.

Port, Sherry, Marsala, Madeira, brandy, gin, or whiskey, possess no peculiar advantages apart from the alcohol contained in each. Spirits contain from fifty to sixty per cent. of alcohol, Sherry and Port from seventeen to twenty-four per cent., and malt liquors from six to eight per cent. Two fluid ounces of spirit will thus be equal to five or six of wine, and spirits ought to be given diluted; and if the prostration is great, and when the skin is cold, and covered with perspiration, the best stimulant is brandy or whiskey punch, given as hot as it can be taken, in small quantities at a time, frequently repeated. In urgent cases stimulants ought to be given every hour; and, as a rule, a larger quantity will be required during the night and early morning than in the daytime, for it is usually towards morning that temperature tends to get low, and the vital powers are at their lowest ebb (MURCHISON). At the same time it must ever be remembered, as Dr. Jenner justly observes, that "in no disease is the advantage of refraining from meddling more clearly displayed than in typhus fever; and in no disease is the prompt use of powerful remedies more clearly indicated. It is in determining when to act, and when to do nothing, that the skill of the physician as a curer of disease, in the case of fever, is shown. Interfere by depletion or by stimulation when nothing should be done, and the patient is lost, who, if it had not been for you, would have been safe. Refrain from depletion or withhold stimulants

when the one or the other is required, and the patient sinks into that grave from which judicious treatment might have saved him."

A large well-ventilated apartment, fresh air, a cool, but not a cold atmosphere, quiet, abstinence from solids, and a free supply of water, milk and water, coffee, weak broth, beef tea, according to the discretion of the physician, are the conditions and remedies on which a large majority of cases will recover. But the patient must be constantly and carefully watched, and there is no disease where the attentions of a well-instructed nurse are more demanded; and there is no class of patients in hospital so apt to be neglected by the attendants, especially as to the regular administration of the remedies prescribed. It is not uncommon to find that the wine allotted for the day has been administered at a draught, when it ought to have been given in small quantities at regular intervals, with care and watchfulness. How often do we see almost hopeless cases recover under the careful nursing of an intelligent person, regulated by the dictates of common sense and conscientious solicitude, guided by the judicious directions of a physician who knows well the nature of the disease with which he has to deal! The nurse ought to note down the hours at which food or medicine has been given, or any remarkable change in the symptoms. She might also, if she were instructed, take observations with the thermometer, for the information of the physician at each visit.

Dr. Murchison recommends that, in urgent cases, food and alcoholic stimulants must be persisted with as long as the patient is able to swallow; and even when he can no longer swallow, the case is not to be given up; for he has seen cases where life appeared to be saved by frequent enemata of beef tea and brandy after the patient had ceased to take anything by the mouth.

Of special symptoms which call for relief, the most urgent is generally headache. If headache should persist after delirium sets in, with a rapid pulse (*e. g.*, 120), attended with nausea, some saline effervescing mixture, with four drops of hydrocyanic acid, may be given every six hours. In the persistence of headache, dry cupping, such as has been recommended by Dr. Sieveking, might furnish an aid to guide the treatment, by determining whether it may not depend upon repletion or upon emptiness of the cranial vessels. When applied to the nape of the neck, dry cupping may afford relief, if repletion has to do with the continuance of headache. Under such circumstances the face is generally flushed, the conjunctivæ red, and the skin dry and hot. If the dry cupping does not relieve such symptoms, the hair must be shaved off the head, and the scalp covered with crushed ice inclosed in a bullock's bladder, or recourse may be had to the cold affusion. The application of cold water is best effected by bringing the patient's head over a basin at the edge of the bed, and having a vessel arranged so that the cold water (at 40° or 50° Fabr.) may drip continuously from a height of two or three feet upon the head (MURCHISON). A skein of worsted arranged in the water, with the ends overhanging the basin, will maintain a constant flow of water from the basin, and which may be directed to fall upon the scalp. Dr. Murchison recommends that in young

subjects two or four leeches may be applied to the temples; and in aged or infirm persons warm fomentations to the head are advisable (GRAVES and MURCHISON). But if anæmia is the cause of the headache, as may be suspected from the state of the vascular system, then stimuli are called for. Four to six ounces of wine may be given in divided doses during the day and night of twenty-four hours. If the pulse continues to get weaker, the wine must be increased.

The headache of typhus naturally abates about the eighth day; but it is sometimes rendered worse by sleeplessness; and if the remedies for the headache do not relieve it, nor tend to induce sleep, then opiates may be given, combined with antimony, if the skin be dry and hot and the pulse of good strength. Dr. Murchison thinks that the employment of opium in typhus is more dreaded than it ought to be. The dose of opium should be given about 9 P.M., followed in two hours by half the dose if the patient does not sleep. The form of the opiate and dose may be ten to twenty minims of Battley's solution of opium, or fifteen minims of the solution of the *bimeconate of morphia*, or five grains of the *opium pill* of the *British Pharmacopæia*. Dr. Murchison teaches us to distinguish two forms of delirium as a guide to the administration of opium, combined with antimony in the one form, and with ethereal stimulants in the other. When the condition of the patient approaches more to that of *delirium ferox*, the cardiac and radial pulses being of good strength, after trying the cold affusion, and remedies already mentioned, then opium combined with antimony ought to be given without delay, as in the following prescription:

R. Liq. Opii. Sedat. (Battley's), ℥℥x; Antim. Tart., gr. j ad gr. ij; Aquæ Camph. ℥vj; *misce*. A large spoonful of this mixture is to be given every hour until sleep is induced.

On the other hand, if the delirium approaches in its character that of *delirium tremens*, the radial pulse is usually quick and feeble, the cardiac impulse diminished, and the first sound of the heart more or less inaudible, then the opium must be combined with alcoholic or other stimulants, the amount being regulated by the state of the pulse and heart. Dr. Murchison suggests the following prescription:

R. Liq. Op. Sed. (Battley's), ℥ss.; Spt. Ætheris, ℥℥x; Aquæ Camph., ad ℥iij; *misce*. Commence by giving two table-spoonfuls of this mixture, and repeat it every hour till sleep is obtained.

Or opium to the amount of half a grain may be combined with three grains of camphor in a pill, and such a pill may be repeated, if necessary, every two hours.

Cases requiring such treatment ought to be seen at least three or four times daily. If dyspnœa is urgent, and lividity of the face betoken pulmonary lesion, defective arterialization of the blood, and venous congestion of the brain, opium in any form must be withheld; and it must likewise be discontinued if any tendency to stupor

supervene, or if there be any marked contraction of the pupil—*e. g.*, “the pin-hole pupil” of Dr. Graves. This physician proposed the use of *belladonna* in such cases, and he, as well as Dr. Benjamin Bell and Dr. Murchison, bear their united testimony to its usefulness. Dr. Graves prescribed it as follows:

R. Ext. *Belladonnæ*, gr. j; Ext. *Hyoscyami*, gr. vj; Pil. *Hydrar.*, gr. xxx; *misce.* This mass being divided into six pills, one may be given every three hours; or it may be given in the form of a draught, in the following prescription: R. Ext. *Belladonnæ*, gr. j; Pulv. *Moschi*, gr. x; *Mucilaginis Acaciæ et Syrup. Aurant.*, āā ʒcxxx; *Aquæ Camph.*, ʒss.; *misce.* A draught of this composition may be given every six hours.

Dr. Murchison considers that musk and camphor are stimulants of very great value, which have fallen into unmerited neglect. Camphor may be given in emulsion in doses of five grains every two hours; or in the form of an enema in doses of a scruple. Huss and Graves also bear testimony to the good effects of these remedies. In a case of complete sleeplessness Dr. Graves gave the following combination of these medicines with the best results:

R. *Antim. Tart.*, gr. ss.; Pulv. *Moschi*, gr. x; Camphor, gr. v; Tinct. *Opii*, ʒx; *Aquæ dil.*, ʒj; *misce.* A similar draught may be given every two hours; and after the third dose the patient will generally fall into a quiet sleep.

When there is danger of stupor passing into profound coma, Dr. Murchison has seen the best effects result from a small cupful of a strong infusion of coffee given every three or four hours, employing at the same time such measures as have a derivative action on the kidneys, *e. g.*, dry cupping; mustard poultices to the loins, wet compresses of thickly folded flannel, wrung out of hot water, passed round the loins, and covered with a piece of waterproof cloth, retained in its place by a bandage or a towel. These remedial agents are all the more necessary if the urine contain either blood or albumen. At the same time free evacuation from the bowels should be secured by a purgative, or by a turpentine enema. If the lethargic state supervenes early, and before there is great exhaustion, the douche has been found to be of great service as a stimulant, provided there be considerable elevation of temperature, and little irritability of the nervous system (TODD, ARMITAGE, MURCHISON).

The region of the bladder should be examined by the physician at least two or three times daily, by manipulation and percussion; and if there be the slightest doubt as to its containing urine, the catheter must be introduced.

Origin and Propagation of Typhus Fever.—It is yet uncertain whether great overcrowding and vitiation of air by the organic impurities emanating from the respiratory and other functions will absolutely generate typhus fever *de novo*. In all English wars (for “typhus fever is a disease as old as the disputes of nations”) there has always been plenty of typhus poison waiting for favorable conditions to assume activity. This arose from the peculiar system of

recruiting. Commissions or commands of regiments were wont to be given to those who collected a certain number of men. Every low purlieu, every infamous haunt, every jail even, used to be ransacked for recruits. Wherever these men went they carried typhus, at that time the constant scourge of our towns and our jails; and complaints of the introduction of typhus fever from this source are frequently found in the writings of army surgeons of the last century. In connection with this point, Dr. Donald Munro, in 1764, gives the following caution: "That particular regard be paid to those soldiers picked up in the streets, or who have been taken out of the Savoy or other jails. All dirty rags from such people should be thrown away or burnt" (DR. PARKES, *l. c.*). There is now ample proof that typhus fever may be communicated *by fomites* adhering to apartments, articles of clothing, and the like; and, provided fresh air be excluded, it is known that such articles will retain the poison for a very long time. Herein lies a fallacy which pervades the argument from cases to prove the generation of the disease *de novo*. The poison may be said (like that of small-pox) to be constantly in existence. Dr. Murchison quotes some striking instances of the propagation of typhus fever by *fomites*. For example, he refers to the instance related by Foderé, in which the soldiers of the French army, during their retreat from Italy in 1799, communicated fever to the inhabitants of towns and villages where they halted on their route, although the army was not attacked by fever, and soldiers travelling *singly* did not communicate the disease. But as he omits to connect this with the fact that typhus prevailed to a great extent in the towns they besieged, and in some instances obtained possession of, the source of the fomites is not made apparent, and therefore in my last edition I was made to misrepresent this instance given by Dr. Murchison, and to put it forth as an example of generation *de novo* (see p. 87 of his work *On Continued Fevers*). He quotes, however, the recent instance of the Egyptian vessel, the "Scheah Gehald," at Liverpool, the crew of which disseminated the poison of typhus by their clothes and persons, although, as he says, they had not the disease themselves. But this is an error. The careful investigation made by Dr. Parkes into the history of this epidemic on board the Egyptian ship clearly shows that the crew suffered from typhus fever (*Statistical, Sanitary, and Medical Reports of the Army Medical Department* for 1860, p. 359). The facts of the case have been curiously confused; but the following statement, from the above and other sources, may be relied on: A number of men (476, chiefly Arabs) were shipped on board the "Scheah Gehald" at Alexandria, to proceed to Liverpool to navigate back a man-of-war then in that port. The weather was cold and stormy, the hatches were battened down during a lengthened voyage of thirty-two days from Malta; and the men, unaccustomed to the rigor of a Northern winter, and not provided with suitable clothing, crowded below for warmth and shelter. Even they whose turn it was for duty had to be driven up on deck. They were extremely crowded on board, and the space below deck was quite insufficient for so large a number (for the crews of two vessels were on board); and there was no at-

tempt to promote ventilation. The persons and clothing of the men were filthy in the extreme. The space between the decks soon became intolerable from filth; for many of the men, being landsmen, were sea-sick on the voyage, and they discharged the contents of their stomachs and bowels in all parts of the ship, which, on arriving at Liverpool, was so offensive that it had to be sunk in the graving dock. Moreover, the rations served to the men were much below the proper standard as regards quantity. Several deaths occurred on the voyage; and although the captain denied the existence of fever and the occurrence of deaths, his statements are quite untrustworthy, for on arrival in Liverpool thirty-two men had to be sent to the Southern Hospital. Two died soon after admission, and their disease was returned as dysentery; but Mr. Pemberton, on whom the duty of receiving and treating the patients at first fell, was convinced that he had some kind of fever before him in the persons of these sick Arabs. He called the disease "febris;" and in writing to a friend expressed his belief that it was a "jail fever." The heat of skin, the sordes on the teeth, and the marked symptoms of stupor in some cases and furious delirium in others, led him to this conclusion, although he could not see the eruptions on the dusky skins of the Egyptians; and perhaps, as he had never seen a case of typhus fever before, he might not have recognized the eruption, as Dr. Parkes suggests, even if present. This diagnosis, however, was made at once by Mr. Pemberton, and before fever had been communicated to any residents in the hospital. It is impossible now to ascertain how many of the thirty-two Egyptians had this fever; but five had marked, and several others had slighter symptoms. Many of the patients (typhus and others) had dysentery, and several were frost-bitten.

Indubitable typhus fever, with a well-marked rash, was communicated by these men, and by Mr. Pemberton himself (who had a well-marked rash), to another medical officer, and to two nurses, a porter, and some patients. The chaplain also, who slept out of the hospital, but visited the sick men, was attacked and died in twelve days. In all, nineteen persons contracted typhus in the Southern Hospital, three on board the ship after she came to Liverpool, and three at the Liverpool baths. Six died of these twenty-five persons. "No single link of evidence," says Dr. Parkes, "is wanting here to show that typhus prevailed on board the ship, and that typhus patients admitted from the ship into hospital communicated the disease to a number of other persons. The idea that the Egyptians suffered only from dysentery, and that in some remarkable way a specific disease like typhus arose out of this dysentery, does not appear to have the slightest foundation. To urge such an hypothesis, in the face of the simple facts above noted, is to ignore all evidence, and to render the progress of medical science impossible. Cases of typhus were not only communicated to residents in the hospital, but to persons who boarded the ship, and to three attendants at the public baths, to which more than 200 of the crew were sent. Some of these men were sick, though they were not known to have typhus. They carried typhus, however, in some way—per-

haps in their clothes—and communicated it to the attendants. The remaining crew (350) of the 'Scheah Gehald' were sent to Alexandria on board the 'Voyageur de la Mer.' The people of Liverpool were probably so glad to get rid of them that they did not take the trouble to see that the typhus fever had been eradicated, and several of the men were sent at once from the Southern Hospital. The 'Voyageur de la Mer' lost some men on the passage, and landed several at Falmouth, and some with unequivocal typhus at Malta; and of thirteen Englishmen who were in her, six took the disease."

"The case of this Egyptian vessel," continues Dr. Parkes, "afforded almost the best opportunity seen in this generation for the investigation of the important question of the spontaneous generation of typhus. The opportunity was, however, lost. That all the circumstances which have been supposed to be capable of calling into existence the specific poison of typhus were present in this foul and filthy ship is clear; but every one who reads all the published statements will at once perceive that one link of the chain of evidence is wanting, and that it has *not* been proved that some of the crew were not ill with typhus when they embarked at Alexandria, or became ill within the incubative period. On the contrary, the interpreter informed Mr. Pemberton that some of the men were sick when they came on board. It can never now be ascertained whether there were such cases or not, and the history of the outbreak at Liverpool affords another instance of the loss of a great opportunity for definitely setting at rest a most important question." The case of the "Scheah Gehald" now assumes exactly the same aspect as many instances historically quoted as examples of generation *de novo*—namely, that however plausible may seem the probability, there is *no proof* that typhus fever arose *de novo*. Seeing that such is the state of the question as to the origin of typhus—that it is exactly in the same state as our knowledge regarding the origin of small-pox or of typhoid fever—that it has been in existence from the earliest periods of the world's history—that it is undoubtedly propagated from pre-existing *fovi*, and by continuous succession, the immediate direction of investigation ought to bear especially on the following points, namely: How long can the typhus poison exist or be maintained in a condition fit to assume activity under favorable circumstances? What is the distance at which it is potent? Has temperature any influence upon it? What are the conditions or combination of circumstances more or less essential to the development and propagation of the typhus poison.

The fact that typhus fever is contagious is based on evidence which shows,—(1.) That, when typhus commences in a house or district, it often spreads with great rapidity; (2.) That the prevalence of typhus in single houses, or in circumscribed districts, is in direct proportion to the degree of intercourse between the healthy and the sick; (3.) That persons in comfortable circumstances, and living in localities where the disease is unknown, are attacked on visiting infected persons at a distance; (4.) That typhus is often imported by infected persons into localities previously free from it; (lastly), That its contagious nature is indicated from the success

attending the measures taken to prevent its propagation, more especially the early removal of the sick. Dr. Murchison fully illustrates by examples all of these statements.

The specific poison seems capable of transmission in various ways; but many circumstances seem to point to the cutaneous and pulmonary exhalations of the sick as the media which convey the specific poison from the diseased to the healthy. It is thus conveyed through the air, or by fomites. That particles of organic matter are constantly floating in the air no one can doubt who has seen the ingenious contrivance of M. Pouchet put in practice, and the substances so suspended in the atmosphere collected by drawing a current of air through a funnel with a very small opening. Immediately below the opening the covering glass of a microscope slide is placed, with a drop of glycerine spread over it. Upon this the current of air impinges, and any solid or corpuscular bodies floating in the air may be in this way arrested and examined with the microscope. Dr. Parkes has detected, by this method, unequivocal epithelium-cells in several instances; and Eiselt, in a ward containing thirty-three children with acute purulent ophthalmia, was able to detect pus-cells floating in the air (*Army Med. Dep. Sanit. Report* for 1860, p. 346; also *Comptes Rend.*, 1861; *Med. Times and Gazette*, 1861).

Such material particles, capable of conveying the specific poison of a disease such as typhus, are thus inhaled or swallowed, and so they find admission into the bodies of the healthy, to exercise their morbid influence on the blood. A peculiar pungent odor emanates from the typhus fever patient. It is especially obvious from the breath, and from the skin on turning down the bed-clothes.

There is no evidence to show the extent of space through which the typhus poison can be transmitted through the air. From some observations it would seem that the contagious influence of typhus is confined to a narrower sphere than that of small-pox. Dr. Murchison concludes that, "if a patient be placed in a large, well-ventilated apartment, the attendants incur little risk, and the other residents in the same house none whatever.

"There are likewise no grounds for the popular belief that typhus may be propagated through the atmosphere from a fever hospital to the houses in its neighborhood. On the other hand, medical attendants who auscultate typhus patients, or who inhale their concentrated exhalations from under the bed-clothes, run no small danger, and the danger is always increased or diminished in proportion to the supply of fresh air" (MURCHISON, *l. c.*, p. 80). There are also good grounds for believing that typhus fever may be communicated, and even carried a great way, by fomites, or by articles of clothing strongly impregnated with the specific poison; and, provided fresh air be excluded, the efficiency of the poison may be maintained for a long time. "Complaints of the introduction of typhus from this source are frequently found in the writings of army surgeons of the last century. Typhus was several times carried to the West Indies, and even there prevailed apparently to some extent" (Parkes *On the Causes of Sickness in English Wars*). The

poison may also adhere to the walls of dwellings, to beams of wood, and to articles of furniture. Dr. Murchison quotes an account by Pringle of twenty-three persons being employed in refitting old tents in which typhus patients had lain; and seventeen of these persons died of the infection. He also refers to an observation of Lind, who mentions several instances in which infected ships continued to impart the disease long after the sick had been removed. Similar cases are recorded by Jacquot respecting the Crimean typhus.

Nurses and other attendants in fever hospitals are well aware of the danger of contracting typhus from infected clothes, and from cleaning the bedding of the sick; and in some instances they are in the habit of "measuring the amount of danger by the badness of the smell." Thus they are liable to contract typhus fever without having had any direct communication with the sick. With regard to the kind of clothing most apt to retain and convey the specific poison, woollen textures are found to be the most dangerous. Haller, of Vienna, has made experiments on this point. He observes that *dark-colored* materials of clothing are more apt to absorb the contagion of typhus, and to convey it to other individuals, than those which are light-colored. He found that, among troops wearing dark-colored uniforms, it more frequently happened that new cases of typhus entered the hospital after a convalescent patient joined his corps, than those wearing light or white uniforms. The fact has been often observed, that in dissecting-rooms dark clothes acquired the cadaveric odor sooner, and were deprived of it less readily than light ones; and he ascertained by experiments that the absorption of odors is regulated by the laws which govern the absorption of light. Haller also found that the specific poison of typhus fever is lighter than atmospheric air. When the under stories of an hospital were filled with typhus patients, those in the upper stories were always observed to become infected when there was a communication between the air of the two stories. On the other hand, when only the upper stories contained cases of typhus, the patients in the under part of the house enjoyed perfect immunity (*Edin. Med. and Surg. Journal*, 1853). Dr. Murchison has observed that, if the poison be very concentrated, the length of the period of exposure sufficient to contract the disease is very brief—not more than a few minutes; and the latent period during which it remains in the body, without betraying its presence in any way, has been very variously estimated. Nine days is the result of Dr. Murchison's observations. Instances, however, are not uncommon in which the disease manifests itself almost instantaneously after exposure to the poison. In such cases these extremely susceptible persons are generally conscious of the peculiar and offensive pungent odor emanating from the beds or bodies of the sick. They are generally then immediately seized with prostration, nausea, rigors, and headache, followed by the regular development of the disease. Such persons are thus almost conscious of the moment at which the poison entered their system. On the other hand, the length of time between exposure and attack may be greatly prolonged. In my own case, I was three months in daily and close attendance in the fever

wards of the Dundee Infirmary, for many hours, on cases of typhus fever, before taking the disease. In such cases, however, of prolonged exposure, it is probable that the constitution may be susceptible at some periods rather than at others; but of the conditions of such susceptibility nothing is known.

Opinions vary as to the stage of the disease at which the typhus poison is most powerful. Some consider that it is so during the period of the eruption—others that it is so during the period of convalescence. Dr. Perry was of this latter opinion, and Dr. Murchison's observations lead him to confirm the opinion of Perry; but he is inclined to think that the disease is really most apt to propagate itself from the end of the first week up to convalescence, when the peculiar odor from the skin and lungs is the strongest.

The conditions essential to the propagation of the specific poison of typhus fever are mainly as follows: (1.) Overcrowding, coexisting with deficient ventilation; (2.) Personal squalor, and filthy apparel saturated with cutaneous exhalations; (3.) A deteriorated state of the constitution, such as may result from protracted starvation, scurvy, and other debilitating causes; (4.) A moderate temperature. Dry heat is a powerful disinfectant.

RELAPSING FEVER.

LATIN, *Febris recidiva*; FRENCH, *Typhoïde à rechutes*; GERMAN, *Typhus recurrens*; ITALIAN, *Tifo ricadente*.

Definition.—A continued fever, having a very abrupt invasion and short duration, marked by rigors, chilliness, and severe headache, vomiting and often jaundice; a white moist tongue, epigastric tenderness, confined bowels, enlarged liver and spleen, high-colored urine, a frequent, full, and often bounding pulse, pains in the back and limbs, restlessness, and occasionally delirium. These symptoms abruptly terminate by an exceedingly copious perspiration between the fifth and the eighth day; and after a complete apyretic interval (during which the patient may be so well as to get up and walk about), an abrupt relapse supervenes on the FOURTEENTH day from the first commencement. The relapse runs a similar course to that of the primary paroxysm, and terminates between the third and the eighth day. In some cases a second, third, fourth, and even fifth relapse may occur. Death is apt to happen from sudden syncope, especially after the excessive perspiration; or from suppression of urine and coma. No constant eruption and no specific lesion are associated with this fever.

Pathology.—The name by which this disease is known is derived from one of the most constant and striking peculiarities of the fever. It has been also described under the various names of "five" or "seven-day fever," "seventeen-day fever," "bilious remittent fever," and "bilious relapsing fever," "mild yellow fever," "synocha," "short fever," and "short relapsing fever." Epidemics of this form of fever have been recognized to prevail on different occasions since 1739. In Dublin it prevailed at that time and in several subsequent years.

Sometimes it has been described as a variety of a well-known form of fever, and at other times as a new disease.

In Scotland in 1817-18 this fever was clinically recognized and described by Drs. Christison and Welsh; and when it reappeared as an epidemic in Edinburgh and Leith in 1843, Dr. Christison had no difficulty in again recognizing it. About this time it also appeared in Glasgow as an epidemic about a month before its outbreak in Edinburgh; and subsequently it became prevalent in Dundee and other large towns in Scotland. It was observed with great accuracy, and its phenomena were recorded in the medical journals of the period, by Drs. Craigie, Alison, Arrott, Henderson, Douglas, Jackson, Mackenzie, Cormack, and Wardell. It formed a part of the fever epidemic of Ireland in 1817-18-19, described by Barker and Cheyne; and it had been prevalent in Ireland for many years. Epidemics of it were described by Ratty, in his *Chronological History of the Diseases of Dublin*, as early as 1739 and 1741. In most of the periods of epidemic fever referred to, the commencement of the epidemic was characterized by the greater preponderance of cases of relapsing fever; and as the epidemic advanced, the number of cases of relapsing fever gave place to a preponderance of typhus cases (STEELE, R. PATERSON, ORMEROD, MURCHISON). In 1847 it became again epidemic in Glasgow, Edinburgh, and the large manufacturing towns of Scotland, as well as in London, when it was carefully described by Dr. Jenner, who, moreover, shows that its characters have remained constant since they were first described by British physicians. During the same year it prevailed in some parts of the Continent, and more especially in the Prussian province of Upper Silesia, and in some other parts of Germany. There it has been described by Virchow, Bärensprung, Dümmler, and Suchanek. These observers, however, did not know or recognize the fever so well and precisely described by the Scotch physicians; and, indeed, Dr. Parkes, was the first to indicate, in his admirable paper on "The Diagnosis of Fevers," already noticed, that the epidemics these German physicians described were mainly made up of the relapsing fever. This fever evidently formed the great bulk of the cases. Yet, although its characters are thus so striking that the most superficial observer could not fail to recognize them, the German systematic writers (except Virchow) make no allusion to relapsing fever as a separate and distinct disease; and even those who observed the fever in Germany failed to draw that obvious inference to which the Scotch physicians unanimously came—namely, that *relapsing fever* is a disease altogether distinct from *typhus* and from *typhoid fever*. If it is not so, "we know not," as Dr. Parkes observes, "that any medical evidence whatever can be relied upon."

In the summer of 1855 it prevailed, after the hardships and privations of the preceding winter, among the British troops in the Crimea, where it was recognized and described by Dr. Lyons. It has not been observed in France, nor in any other part of the continent of Europe.

The observations of Dubois, Austin Flint, and others, leave no

doubt that relapsing fever was seen in New York, Buffalo, and other parts of North America, in 1847 and 1848; but Dr. Murchison is of opinion that all the cases are traceable to Irish immigrants, and that there is no good ground for believing that the disease is indigenous in America. It has been well described by Dr. Wood, of Philadelphia, from the writings of the physicians already mentioned.

[The first cases of relapsing fever observed and reported in this country were by the writer.* In June, 1844, a vessel, with Irish emigrants, arrived from Liverpool at Philadelphia. There had been some sickness in the steerage during the passage. On her arrival fifteen of the passengers were admitted into the Philadelphia Hospital, suffering from a form of fever identical with that which was then prevailing in Scotland and parts of England, and described by Alison, Cormack, and Henderson—the same ardent fever, bronzed hue, profuse sweating, sudden intermittence from the sixth to the seventh day, steady improvement for five or seven days, and sudden relapse, sometimes severer, sometimes milder than the initial attack. In no case were the sequelæ observed; but these might have occurred, as the patients left the hospital on the establishment of the second convalescence. None of the cases ended fatally. The disease, so far as the writer knows, did not extend; the only evidence of its communicability that came to his knowledge was the cases of two sisters admitted into the hospital with the fever, who had been residents of the city for several years, but whose brother, a passenger in the ship, had been attacked with the disease at their house. Several of the passengers were also inmates of his hospital, suffering from slight chills and sweatings, headache, nausea, and loss of strength, but recovered without a fully developed attack. Several cases from the same vessel were admitted into the Pennsylvania Hospital, under the charge of the late Professor Pepper, who at first regarded them as cases of mild typhus, but subsequently agreed with the writer as to the real form of the fever.

Towards the end of 1864, and the first part of 1865, relapsing fever made its appearance at St. Petersburg, and, not being at first recognized, excited much alarm, and was for awhile thought to be the Oriental plague. The analogy of its symptoms and course with the relapsing fever of Great Britain leaves no doubt of identity. The mortality in the St. Petersburg fever was 8 per cent., death generally happening in the relapse, and was often preceded by symptoms of general paralysis, much disorder of the nervous system, decomposition of the blood, enlargement of the spleen and liver, &c., without any trace of the intestinal lesion of typhoid fever. Doubowitski, in *A Report to the English Ambassador at St. Petersburg by the Russian Government*, states that, though it occurred for the first time at St. Petersburg in 1864-65, it had prevailed at Moscow in 1840, and in 1858 at New Archangel in Russian America. Bernstein gives an excellent history of an epidemic at Odessa in 1863-64-65. The mortality was insignificant. The Russian epidemic has been particularly described by Tillner, Fetterman, and Ruttner (*Gaz. Méd.*, 1865; *Gaz. des Hôp.*, 1865; *Deutsche Klinik*, 1865; *Rep. of Med. Officer of the Privy Council*, 1865.)

Dr. Baldon has published an account of an epidemic of relapsing fever

* Fevers; their Diagnosis, Pathology, and Treatment. By Meredith Clymer, M.D. Philadelphia, 1846, p. 99.

which he saw in South America. It broke out in Peru in 1854, and proceeded along the chain of the Andes, never at a lower elevation than 1500 metres, until in 1859 it had reached Bolivia and the neighboring parts of Chili. In about 1000 cases the deaths were 250, equally divided among both sexes—children under 16, 92; adults, 110; over 60, 48 (*Gazette Médicale*, 1865).]

In India and in all tropical countries it is as yet unknown.

Since the epidemic of 1847 and 1848, Dr. Murchison writes that relapsing fever has been gradually disappearing; and for the seven or eight years previous to 1863 not one case has been observed in the hospitals of Edinburgh, Glasgow, or London. Professor W. T. Gairdner has not seen or heard of a single case at Edinburgh since 1855; and, according to Drs. Lyons and McEwen, true relapsing fever has of late years been a rare disease in Ireland.

Like other continued fevers, its specific cause is unknown; but it selects its victims from the poor and ill-fed, who live miserably, in crowded, filthy, ill-ventilated apartments, rather than from the wealthy and well-fed, who live in comfort and in well-aired abodes. Its poison appears to be of a specific kind, and the phenomena of the fever are very different from those of *typhus* and *typhoid fever*. Patients recovering from either *typhus fever* or *typhoid fever* may catch, by contagion, the *relapsing fever*, while patients convalescent from *relapsing fever* may also take either of the forms of *continued fever* already described. It has been supposed by some (Dr. COR-MACK) to be identical with the malarious form of *yellow fever*; but there is not sufficient evidence to establish the point. It seems more nearly to approach in its nature some forms of remittent fever, on account of the repetition of the rigors, often at regular daily periods, for two or three days. The marked periodicity of its relapses, which "come on like a fit of ague almost to an hour" (Dr. R. PATERSON), and the enlargement of the spleen to a greater extent than in any other form of fever (JENNER), point also to a malarious origin. On the other hand, epidemics of relapsing fever, as Murchison shows, appear to commence, progress, and decline quite irrespectively of the season of the year.

The evidence that a specific poison exists and is formed in cases of relapsing fever, and when so formed is communicable from the sick to the healthy, rests on evidence similar to that adduced in cases of typhus; and the same objections may be taken to the evidence which aims at establishing the spontaneous generation of the specific poison. There are causes, circumstances, or conditions which obviously favor the accession of relapsing fever, and no doubt also, its occurrence in an epidemic form; and chief amongst these predisposing causes must be placed destitution and want of food, while the names applied to the disease by different countries indicate the popular belief as to such predisposing causes being credited with originating the disease in the first instance. Thus it is spoken of as the *famine fever* of the British Isles, and the *hunger pest* of Germany.

The Primary Paroxysm.—The seizure is generally, indeed almost

always, sudden. Sometimes, on waking in the morning, or when employed in business, severe rigors at once come on, with a sense of chilliness and frontal headache. These phenomena are more severe than their expression is in the commencement of typhus. There is slight prostration of strength from the first, but rarely so severe as in typhus. If premonitory symptoms exist, they usually manifest themselves by pains in the limbs and lassitude, nausea, and perhaps vomiting, with feeling of prostration. Subsequently, and very soon, febrile reaction sets in, sometimes violent, expressed by intense heat of skin, severe headache, throbbing temples, intolerance of light and sound, suffusion of face, sleeplessness, remarkable anxiety of countenance and jactitation, with a very rapid pulse—so rapid as to range from 110 sometimes as high as 140 beats in a minute; the tongue is coated with a white fur, and in a great majority of cases, in some epidemics, there is uncontrollable vomiting of greenish, bitter fluid, with or without epigastric tenderness, and great thirst. The pains in the muscles and joints are sometimes so severe as to resemble rheumatism, and when the pain in the back is severe, together with the rigors, the vomiting, and the headache, it may not be possible in the first instance to say that the attack may not prove to be one of small-pox. But the pain in the back is not generally so severe, nor is the vomiting so incessant in cases of *relapsing fever* as in cases of *small-pox*. The headache is to be distinguished from what is commonly called a "sick headache" or "bilious headache," by the circumstance that the "bilious headache" is in most cases *occipital*, and the heat of skin, combined with the quick pulse, serve to distinguish an attack of relapsing fever at its outset from one of "bilious headache." From idiopathic head affections the accession of relapsing fever is distinguished by the suddenness of the attack, the rigors, the hot skin, the pain in the joints and limbs, and the white tongue (Dr. JENNER). The symptoms generally of *relapsing fever* are so severe that the patient takes alarm, and takes to bed at once. He does not feel weak, but he feels so giddy that he is unable to remain out of bed, or off the horizontal position. In some cases there is pleurodynia in a severe degree, but without any stethoscopic indications of pleural inflammation.

By the second or third day the pulse almost invariably exceeds 100; as a rule it reaches 120; in not a few cases it is as high as 140 or 160; and it is not rarely 140 on the second day of the disease, being at the same time full, and of considerable firmness—symptoms not indicative of commensurate danger—with anxious and oppressed breathing. There may be also sweating, profuse and lasting for several hours, but without relief to the headache and other symptoms. Almost no sleep is obtained, and the little obtained is dreamy and unrefreshing. The skin continues dry after the sweating ceases; or after the primary rigors, if sweating has not taken place; and the heat of skin is ardent—as much as 102° to 107° Fahr.; and these febrile phenomena are occasionally varied by short rigors or slight sweating. Delirium does not generally supervene on the first attack, although, by the *fifth* or *sixth* day, just before the crisis, it has been in some cases of a violent kind. In a

large proportion of cases there is decided jaundice, and in others the skin exhibits a bronzed hue. The jaundice is not attributable to any obstruction of the *ductus communis choledochus*, as bile passes freely, and even copiously, with the stools, and as, after death, the gall-duct is pervious. There is generally tenderness over the region of the liver in such cases; and it may be enlarged. Thirst is excessive; the appetite absent or voracious, and the bowels constipated. The tongue, at first moist, is covered with a white or yellow fur, which it may retain throughout the illness; and, in many cases, it may become dry all over, or with a brown dry streak down the centre, after the third or fourth day.

The Crisis.—After the patient has continued in this state for a period varying from five to eight days, a sudden change takes place, immediately preceded, in most cases, by an exacerbation of all the symptoms. "When every symptom appears hourly becoming graver—when the restlessness and general distress have reached their highest point—then ensues a most remarkable series of phenomena, followed by a remarkable intermission of all the symptoms, and an apparent restoration to health." This period has received the name of "Crisis," and supervenes generally on or about the *seventh* day, and its advent is rarely prolonged beyond the *eighth*. This change is ushered in by a most profuse perspiration, in some instances with an eruption of miliary vesicles, which breaks out from the whole surface of the skin, and in the course of a few hours the patient appears nearly well. More rarely the change is indicated by epistaxis as well as by perspiration, or by profuse diarrhœa, catamenial discharge, or hemorrhage from the bowels; and after either or all of these apparently critical changes have been established for a few hours, there is a complete and abrupt cessation of all the bad symptoms. The pulse quickly regains the natural standard, the tongue cleans, the appetite and sleep return, and the countenance resumes its tranquillity. This alteration is very often effected within a few hours, and on the following day the patient generally considers himself in all respects quite well, and may so continue to improve rapidly for *four* or *five* days. During this period, however, there are some patients who suffer from violent muscular pains in the limbs.

The Relapse or Recurrent Paroxysm.—About *seven* days after this critical change, or between about the *twelfth* to the *twentieth* day from the commencement of the illness, but generally on the *fourteenth* day, a sudden relapse occurs "in ninety-nine cases out of every hundred." This relapse commences suddenly, like the first seizure, by rigors, headache, loss of appetite, vomiting of green fluid, which is quickly followed by a hot skin, quick pulse, and a coated white tongue, confined bowels, followed by delirium, so that the phenomena may be exactly represented as a repetition of the first attack. In the interval of convalescence between the first and second attacks the pulse often becomes slow to an extreme degree, as slow even as forty-five to sixty beats in the minute; but, suddenly, on the relapse commencing, it again rises to 120 or more. In ordinary favorable cases perspiration would again occur in *two*, *three*, *four*, or

five days, and the patient would be relieved as before. The chemical qualities of the sweat have never been determined in cases of relapsing fever; but it has a very sour and peculiar smell. In other cases, however, uncontrollable vomiting, great thirst, very rapid pulse, a hectic-looking circumscribed flush of countenance, jaundice, watchfulness, delirium, and death, may terminate the case.

In some cases the relapse is very slightly marked, and indicated merely by a comparative increase in the rapidity of the pulse and a greater heat of skin, than were present on the previous day. The duration of the relapse varies from a few hours to several days; the average being from *three to five* days, or less than that of the primary paroxysm. In some cases the relapse lasts less than *twenty-four hours*; and in a few it is prolonged to *seven or eight days* (MURCHISON). The relapse is rarely prolonged beyond these periods in uncomplicated cases, but Dr. Lyons observed in the Crimea that the fever of the relapse was occasionally protracted to *twenty-one days* (Lyons *On Fever*).

If blood be taken from the arm, it is generally buffed, but it is *not* to be argued that *therefore* the lancet must be used in *relapsing fever*. So far as can be ascertained, no local inflammation attends it.

In nearly a fourth of the cases, according to Dr. Jenner, jaundice is present, and is sometimes intense. If present during the first attack, it may disappear before the relapse, and not recur; or it may occur only on the relapse: and it is important to notice, that while the jaundice continues, the stools still retain their natural hue, and may even be darker than common, and at the same time the urine may be frequently loaded with bile. Epigastric tenderness is most marked in the cases where vomiting occurs. When pregnant women are attacked with *relapsing fever* they usually abort, sometimes in the first paroxysm, but often in the relapse, and this event renders the prognosis more doubtful.

There is a tendency in *relapsing fever* to the occurrence of sudden death. It may happen by syncope, immediately after the critical periods, when the pulse becomes so very slow. It may also happen during the progress of the case, during either of the severe periods—namely, during the primary attack or during the relapse. It is indicated by a deep dusky hue of the face, lividity of the hands and feet, and a purple marbling of the whole surface. The trunk feels cool, and the hands feel cold, and without suffering any severe pain, or without sustaining any sudden discharge of fluids, a state of collapse insidiously comes on, from which the patient is unable to be roused, and death may follow in a few hours, generally from *twelve to twenty-four*, even after it was supposed that danger had been escaped. But death is a rare termination to relapsing fever; and when it does occur, the fatal event more commonly happens during the primary fever than during the relapse (DR. JENNER).

A second relapse, and a third, a fourth, and even a fifth, are reported to have occurred during epidemics of *relapsing fever*, but the cases are of rare occurrence.

Duration of the Fever, and Convalescence.—Under ordinary circumstances, when there are but two paroxysms—*i. e.*, one primary

paroxysm and one relapse—the total duration of the fever extends to about *three weeks*; and the convalescence is very slow—much slower than in typhus. The *relapsing fever* is very exhausting in its effects upon the constitution; and, dating the period of convalescence from the termination of the last attack, the time taken to recover is in most cases unusually long. To those, indeed, who suffer from more than one relapse, it is almost impossible to have health completely restored for a long time. They become a prey to various sequelæ of fever, or they continue sickly for months, with pallid countenances, puffed ankles, palpitations, extreme debility, noises in the ears, dimness of vision, diarrhœa, or dysentery. Dysuria is a frequent complication amongst women during the relapse. In many instances during the epidemic of 1847 and 1848 in Ireland, convulsions occurred in cases which otherwise seemed to be progressing favorably, and death invariably followed them. Dr. William Robertson observed in Edinburgh (and the Irish physicians record a similar observation) that delirium of a violent character occurred during convalescence, or after the critical discharge had taken place. It generally came on suddenly, with incessant talking, a rapid weak pulse, followed by perfect unconsciousness, flushed face, and contracted pupil.

[**Anatomical Characters**].—No special anatomical lesion has been pointed out as peculiar to *relapsing fever*. The most constant lesion is enlargement of the spleen, the size attained by that organ being on the whole larger than in either typhus or typhoid fevers. Dr. Jenner has recorded the weight in one case to have been as much as thirty-eight ounces, and of a size in proportion. Its substance is generally softened, sometimes diffuent. It is usually seen at its largest size when death occurs during the final paroxysm; but if death occurs during convalescence, the spleen is of a normal size. Occasionally pale, red, fibrinous infarctions are found in its substance and near its surface. They are easily broken down, have a fine granular fracture, and are considerably firmer than the surrounding tissue, from which they are separated by a distinct line of demarcation. As a rule, there is but little congestion of the lungs, the weights of which contrast singularly with the weights of organs in subjects dead of typhus fever.

The blood in a few cases has been found fluid throughout the body after death; but generally, when drawn from the body during the febrile paroxysm, it is buffed; and decolorized coagula are found in the heart and large vessels after death more frequently than in cases of typhus. In several cases urea has been detected in the blood in considerable quantity. The proportion of white corpuscles is increased, a fact of interest in connection with enlargement of the spleen, and the state of anæmia so commonly observed (CORMACK, ALLEN THOMSON, MURCHISON). The liver is generally large, and the gall-bladder filled with dark thick bile.

Sequelæ of Relapsing Fever.—One of the most common results is the occurrence of excessive pains in the limbs, more especially expressed about the knee and ankle joints; and even the long bones appear to be the seat of these pains in some cases. Combined with

those local pains the joints may swell; and the kidneys are in danger of being implicated. In some respects, therefore, the dangers are similar to those which attend scarlatina. The lymphatic glands are also liable to swell, and so is the parotid gland. Anasarca and furunculi may likewise supervene.

The most important of all the sequelæ, however, is a remarkable affection of the eyes—a form of ophthalmitis—which Dr. Mackenzie first described under the name of "*post-febrile ophthalmitis*." It may occur during the course of the fever, but more often during convalescence, and even some months after convalescence has been established. It was very common in Glasgow after the epidemic of 1843; and assumed two different forms, namely,—(1.) An active inflammation of the shell of the eyeball and of the iris; (2.) An amaurotic state due to congestion of the choroid and the retina (DR. ANDREW ANDERSON.)

These two forms of disease, Dr. Anderson observes, may be associated with two characteristics of the fever itself—namely, "the tendencies to visceral congestions, and to rheumatic-like pains;" while the constitutional character of the ophthalmia is in many cases proved by the unhealthy aspect of the blood, which flows dark, in some cases almost tarry, from the vein. Bleeding is found to be the most effectual—the only effectual—mode of cutting short this dangerous ophthalmia; and a very small loss of blood is found to be sufficient. This is especially noticeable because, during convalescence, tonics and quinine are most likely to be thought of.

Treatment.—All physicians agree that in the primary attack little medicine is required after opening the bowels by castor oil; or by five grains of the compound colocynth mass; or by two grains of blue pill, and three grains of extract of hyoscyamus given at night, and followed in the morning by two drachms of the sulphate of magnesia in compound infusion of roses (MURCHISON). The symptoms are not readily under the control of remedies; the vomiting is often especially persistent. Five grains of calomel, with one grain of opium, has been found more efficient in subduing the severity of this symptom than counter-irritation or effervescing draughts. The violence of the headache in well-fed or otherwise healthy patients, is best subdued by leeches or cupping; and in the poor, weakly, and ill-fed, by blisters to the nape of the neck, or by dry cupping there. Till the crisis comes, the symptoms may be mitigated, but not altogether relieved, and cases of ordinary severity are better left to nature, without interference on the part of the physician. Active purging is to be avoided; and the action of the kidneys is to be kept up by the frequent use of small doses of nitre (ROSS, HENDERSON, CORMACK, WARDELL, MURCHISON). "By keeping up the action of the kidneys from the first," Dr. Murchison justly entertains the hope that we may "prevent the occurrence of uræmic intoxication, which is one of the main causes of death in uncomplicated cases." He recommends the administration of the nitre as follows:

From one to two drachms of nitre are to be dissolved in two pints of barley-water, acidulated with a drachm of dilute nitric acid, and sweetened

with a little syrup. This quantity is to be used up during the twenty-four hours. Acetate of potash and nitric ether may be used for the same purpose; but the nitre has the additional advantage of keeping open the bowels.

The surface of the body should be frequently sponged over with cold or tepid water; stimulants are not usually necessary, but they may be required in the stage of languor or exhaustion ensuing on the crisis; or in cases where great debility has preceded the attack. If any anæmia exists, or if an anæmic murmur can be detected, stimulants must be given early. When jaundice appears, Dr. Murchison recommends that nitro-hydrochloric acid should be given in combination with nitre, as in the following formula:

Twenty minims of hydrochloric acid, with ten minims of nitric acid, every three hours, each dose diluted with the drink of nitre and barley-water already prescribed.

Contamination of the blood with urinary products is the great danger in cases of relapsing fever; and therefore, in all cases of relapsing fever, particular attention must be paid to the state of the urine, especially towards the period of the first crisis. When the daily amount is much reduced, or if entire suppression should ensue, and particularly if stupor, confusion of thought, or drowsiness should supervene, the bowels are to be freely moved by compound jalap powder, or by a turpentine enema. Determination to the skin should be promoted by the hot air bath; and saline diuretics may be given every two or three hours (MURCHISON). No means hitherto discovered will prevent the occurrence of the relapse.

FEBRICULA.

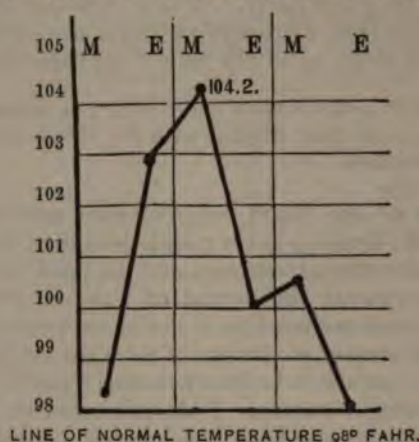
LATIN, *Febricula*; FRENCH, *Fièvre éphémère*; GERMAN, *Febricula*; ITALIAN, *Febbricola*.

Definition.—*A simple fever in which the expression of the febrile phenomena is of very short duration, lasting, as a rule, for twenty-four, thirty-six, forty-eight, or seventy-two hours or more, attended with a frequent, full, and often firm pulse, white and coated tongue, pains in the loins and limbs, thirst, constipation, a scanty discharge of high-colored urine, hot and dry skin, sometimes an eruption of roseola or erythema about the loins or thighs, coming and disappearing with the fever (MOREHEAD); severe headache, sometimes acute delirium, and flushed face. The subsidence of the fever is generally associated with copious perspirations, or herpetic eruptions.*

Pathology.—We do not know of any specific poison as the cause of such phenomena as those detailed in the definition; neither have we any evidence that febricula is a contagious or miasmatic disease. There are many different causes which are known to be capable of exciting expressions of febrile phenomena similar to those mentioned in the definition,—such as exposure to great heat or cold, surfeit, inebriety, mental or bodily fatigue or excitement; and specific poi-

sons, in uncertain or otherwise mild doses, such as the typhus or enteric fever poisons. It is always associated with local and functional disturbances—*e. g.*, catarrhs (bronchial, gastric, intestinal, urethral), milk fever, the fever of alcoholism. Such cases are especially characterized by the apparent severity of the febrile state, the shortness of its course, and the absence of any local complication or specific eruption.

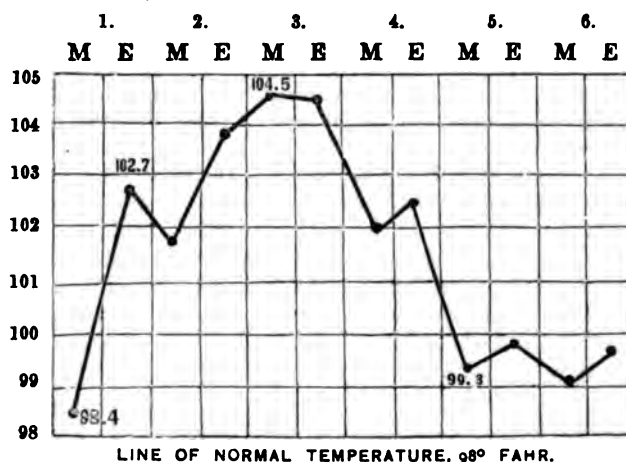
TYPICAL RANGE OF TEMPERATURE IN A CASE OF FEBRICULA. THE RECORDS INDICATE MORNING (M.) AND EVENING (E.) OBSERVATIONS (Wunderlich.)



The pathology of such apparently simple fevers demands extensive investigation, and especially in the tropics, where febricula is a very common disease. The *ardent fever*, the *sun fever*, the *common continued fever* of Burmah and India generally, are all names which indicate severe or protracted cases of febricula—cases of fever which “differ in degree rather than in character” (MOREHEAD). They are common in those parts of India which do not experience much of the influence of the monsoon rains. Cases of true febricula commence with chills, followed by reaction, and this by perspiration. They are characterized by a quick and comparatively sudden rise of temperature, as indicated by the preceding diagram—a rise of temperature in a few hours to 4°, or 5°, or 7° above the normal temperature of 98° Fahr. They have thus a sudden beginning and a rapid arrival at a maximum—phenomena which are only shared in by some forms of *malarious fever (intermittent)*, *variola*, *measles*, and *pneumonia*. The defervescence also is characteristic. In febricula the maximum of temperature may only last for a few hours, or a single day, when the defervescence sets in rapidly; so that in twenty-four or thirty-six hours the body will have returned to its normal heat—an example of *pure crisis*. No other febrile disease gives expression to similar phenomena; and the correlation of temperature to the other phenomena, especially to the excretion of urine, is also peculiar. The urine presents, during this disease, the very type of febrile urine. On the second or third day, according to Dr. Parkes's

own observations, the amount of urine is extremely small (twelve to twenty ounces), of very high specific gravity (1035–1037), with the solids and sulphuric acid very much over the average, and the amount of urea large. When the temperature falls, the quantity of urine rapidly augments. The increase of urea and of the solids is not so much, however, as in the height of the more severe and prolonged fevers (Parkes *On the Urine*, p. 243).

TYPICAL RANGE OF TEMPERATURE IN A CASE OF PROTRACTED FEBRICULA (*ephemera protracta*). THE RECORDS INDICATE MORNING (M.) AND EVENING (E.) OBSERVATIONS (Wunderlich).



In cases of more protracted febricula, the early phenomena are similar to the shorter cases, and the protraction is mainly due to the slowness of the defervescence—an example of *lysis*. The phenomena of such defervescence, as indicated by the range of temperature, are shown in the foregoing diagram.

As a rule, these fevers are not serious; but the degree of reaction has always a relation to the state of the constitution, whether sthenic or not (MOREHEAD).

Treatment of Febricula.—Such means as emetics, purgatives, tepid sponging, diaphoretics, and antiphlogistic regimen, are to be employed. In plethoric individuals, where there is much headache and flushing of the face, a moderate general bloodletting, or leeches to the temples, may be expedient, but such remedies are not often necessary (MOREHEAD).

SIMPLE CONTINUED FEVER.

LATIN, *Febris continua simplex*; FRENCH, *Fèvre continue*; GERMAN, ———; ITALIAN, *Febbre continua semplice*.

There are not a few physicians who doubt the occurrence of *simple continued fever* as distinct from the continued fevers already de-

scribed. On the other hand, there can be no doubt that cases of an anomalous or mixed nature do sometimes occur, concerning which a decided diagnosis cannot be given from the general symptoms merely; and the term *continued fever* "has become a refuge for many cases of an uncertain character."

Thus while the four distinct forms of continued fever previously described are capable of being recognized, there can be no doubt that cases of fever do sometimes occur in this country which run a continuous course, having no other specific characters, and which, in many respects, do not seem quite the same as those with which we are now familiar, and which cannot at once be clinically recognized. For instance, in the very interesting investigation into the nature of "*typhus and typhoid fever*," by Dr. Murchison, recorded in the forty-first volume of the *Medico-Chirurgical Transactions*, it is related that about 200 cases are left out of consideration altogether, because they were "doubtful cases," and could not be classed as either *typhus* or *typhoid* cases.

A similar class of "doubtful cases" are seen to occur in places where *yellow fever* and *remittent* fevers occur, and which cannot be classed as either the one or the other form of fever.

Again, in the Mediterranean latitudes, there is a "gastric remittent" fever described, which seems to have many characters in common with some of the forms of continued fever (CRAIGIE, MARSTON).

Wunderlich and Murchison both describe febrile phenomena which are of so anomalous a kind that they refer them to a combination of the poisons of typhus and typhoid fevers, so that the characters of each do not remain distinctive. So likewise Dr. W. T. Gairdner, in stating that of late (from 1853 till 1862) the cases of fever in the Edinburgh Royal Infirmary have not been more than seven or eight cases a month under his notice—including numerous anomalous fevers which have prevailed, and which have sometimes quite overborne the numbers of genuine *typhus* and of *enteric* fever together (*Clinical Medicine*, p. 154). A fever termed "*gastric*" is distinctly described by Dr. Andrew Anderson, of Glasgow, which would also come under this head. The "*bilious remittent*" or "*bilious typhoid*," of Greisinger of Tübingen, is another form of continued fever which requires investigation. He observed it at Damietta, in Egypt. It is probably a malarious fever of a remittent type, or some form of yellow fever. The sudden fall of the pulse from 120 to 75 was not attended by corresponding improvement of the patient, but was the forerunner of severe typhoid symptoms and jaundice. The mortality was equal to 19 per cent.; and quinine was found to be of signal service (MURCHISON).

The works of Morehead and Sir Ranald Martin may also be referred to for various anomalous forms of continued fever; and in which the "ardent continued fever" of India may be quoted as an example of a very serious disease. For an account of this fever the reader is referred to page 164 of Dr. Morehead's work, and page 204 of Sir Ranald Martin's.

It is these "doubtful or anomalous" cases especially which require careful and special methods of investigation. They are of the ut-

most importance to science, for more extended information regarding them will either connect them with forms of the fever between which they seem to stand; or these "doubtful cases" will eventually separate themselves into distinct forms, whose history is still unknown. In such doubtful cases observations regarding the correlation of temperature, the excretions, the succession of phenomena, and general course of the disease, are imperatively demanded.

The poisons of tropical fevers especially require to be carefully studied, and the phenomena of the febrile state which accompanies them embrace some medical problems of the most abstruse nature. Physiological data of an exact kind are now beginning to rise around us, which will give a standpoint for comparison in the study of the phenomena of fever in the tropics. Extremely important observations are being worked out by Dr. Emile Becher, of the Army Medical Department, regarding the influence of tropical climate on the excretions of the urine in relation to the body weight. At great personal sacrifice and denial of self, he has twice undertaken such investigations on his own person in voyages to India, round the Cape of Good Hope. With all such exact information, and the improved physical aids to investigation, it behooves the physician and pathologist to investigate medical problems with the same logical rigor and severity as a chemical or an astronomical theorem demands. On this important point the opinion of one may be especially quoted, whose experience as a teacher of clinical medicine has been great, and whose philosophical investigations into the nature of fever, in particular, command the respect of all. On this point Dr. Parkes thus writes: "The power of observation in medicine is a kind of tact, which ought to be cultivated with the same assiduity as the chemist practises when he learns how to manage his delicate manipulations, or the astronomer when he wields his wondrous tube. In medicine the observation and recording of phenomena have been held to be an easy and trifling task, which any tyro was competent to do. Hence half the error and uncertainty of medicine. Inaccurate, that is, erroneous and incomplete observation, has been the cause that, till within these few years, the fevers of cold countries have been so absolutely uncomprehended, and that the fevers of hot countries are still shrouded in obscurity. The most valuable addition any one could at present make to our knowledge of tropical fevers would be a simple record of all the cases in an epidemic. These cases should be observed with the keen tact of a Chomel, and recorded with the fidelity of a Louis. We want no explanation or word of comment added to them; we want merely the cases. Then, when the numbers are sufficient, we should certainly begin to put order into this chaos. And let not any one who may have the opportunities be deterred from the task by that fallacious, and, we beg to say, most reprehensible argument, with which some people may favor him,—viz., that his cases will be 'tedious,' 'heavy,' and 'unread.' Unread they will be, certainly, by some of the profession, who consider their routine practice as great an effort as their intellect will bear; but read and analyzed, we will venture to say,

they will be by those who think no labor too great if they can fix safely the foundations of medicine, and for whom, if *accurately* reported, no cases can be too long, no observations too minute. Only, before the task is commenced, let the observer feel that his powers are equal to it; and let him bear in mind the example of Louis, who recorded most carefully for a long time, that he might train himself to this duty, and then throwing his probationary cases aside, as too uncertain for use, began to make those remarkable series of observations which have linked his name forever with the greatest improvements in modern medicine,—the employment of a correct method of studying his science" (*Brit. and For. Med.-Chir. Review*, Oct., 1850, p. 435).

[SPECIFIC] YELLOW FEVER.

LATIN, *Febris flava*; FRENCH, *Fièvre jaune*; GERMAN, *Gelbes Fieber*—Syn., *Gelbfieber*; ITALIAN, *Febbre gialla*.

Definition.—A specific malignant fever of a continuous type, occurring, as a rule, only once during life, and propagated by contagion. It is attended by yellowness of the conjunctivæ and skin, delirium, suppression of urine, interstitial hemorrhages, and hemorrhages from the stomach, mouth, nares, and rectum (black vomit, black stools), a slow and, at times, an intermittent pulse. It is limited to very definite geographical limits, never having been known to propagate beyond 48° north latitude, nor without a temperature of 72° Fahr. at least. It has been imported into Lisbon, into St. Nazaire, in the department of the Lower Loire, into Plymouth and Southampton, where cases have run their course and proved fatal. It has been imported and become epidemic as far south as Monte Video. It has occurred as high as 4000 feet above the sea-level (Newcastle, in Jamaica). But, as a rule, it is endemic in low districts on the sea-coast, and rarely occurs above an elevation of 2500 feet above the level of the sea.

Pathology and Causes.—Yellow fever of a specific kind must now be regarded, from an enlightened consideration of its history, as one *sui generis*, and specifically different from the remittent and intermittent fevers, in which the patient may become yellow, or any other form of malarious yellow fever (CULLEN, CHISHOLM, BLANE, WOOD). There are those who believe that these fevers are the same in kind, but various in degree; that certain atmospheric conditions, such as great heat or humidity, acting on a predisposed frame, will produce all the symptoms of the most malignant fever; that the intensest form of yellow fever is but the developed degree of the common bilious derangements peculiar to hot and rainy seasons (TOMMASINI, CLEGHORN, LIND, HUNTER, ALISON, CRAIGIE, MARTIN). It has been held also that a specific non-contagious agent produces a fever which has been called "yellow," but which is totally different from the real yellow fever (ROCHOUX). On looking carefully into the history of yellow fever, on which volumes have been written, the conclusion arrived at seems to be,—(1.) That there is a specific yellow fever, propagated by a contagious virus or poison which multi-

plies itself by its passage through the human system, and which reproduces the same specific true yellow fever. The type of this fever is continuous. Pyrexia, delirium, suppression of urine, black vomit, are the leading symptoms of this fever—the *hæmagastric pestilence*, as it has been also called. (2.) That there are other fevers, and especially severe marsh fevers, in certain geographical limits, which have a close resemblance in symptoms to the contagious and specific yellow fever. So also, it is said, have fevers arising simply from a high temperature acting on an unseasoned subject. On this point my friend and colleague, Dr. Maclean, Professor of Military Medicine, who has had twenty-two years' experience of East Indian fevers, writes me as follows:

"I am now myself a firm convert to the doctrine that yellow fever is specifically distinct from remittent. To this opinion I have come with a full knowledge of the fact that some cases of remittent fever in India closely resemble some of the forms of yellow fever. But of this I am now certain, that the yellow fever of the true yellow fever zone is unknown in India where true malarial fevers abound. There is in true yellow fever, for the most part, an absence of that periodicity which is an unfailing characteristic of true malarial fevers. Then there is the difference so well insisted upon by Blair in true malarial fevers. Men do not pass from recovery to health, as is the case in such a marked degree in yellow fever, after which there is no, or very little, evidence of the existence of any cachexy. Malarial fevers exist and are destructive at a temperature at which yellow fever is at once destroyed. Albuminous urine is almost invariable in yellow fever—only occasional in remittent. There is in yellow fever an unexampled range of hemorrhages; in remittent fever these hemorrhages are often, indeed generally, absent. Quinine has a power over malarial fevers that is beyond the reach of doubt or cavil; the same is not true of yellow fever. Men suffer from malarial fevers again and again; second attacks of yellow fever are, to say the least, rare."

The correctness of the above view of this important question has also received a remarkable illustration in some observations made in Mexico by the Medical Staff of the French Army serving there.

Yellow fever had disappeared from Vera Cruz. In the month of October, in the middle of a sudden augmentation of sickness, several severe cases of a disease like *vomito* appeared. The physicians dreaded a return of yellow fever in its epidemic form, in spite of the relative abatement of temperature and the almost constant prevalence of a northeast wind. These fears were augmented by the disembarkation at this time of a great many fresh troops; also by the suddenness of the invasion of the disease, showing itself in a great many cases by hepatic symptoms, bilious vomiting, fever, prostration, articular pains; the fever being continuous, or at least without sensible remissions, in many cases. But soon many circumstances demonstrated the groundlessness of the fear, and showed that the disease was malarious. These were,—(1.) The quickly recognized efficacy of quinine, which in the month of May, when true yellow fever raged, gave only negative results at the best; (2.) The rapid supervention of splenic enlargement, often attended with

pains; (3.) The small relative mortality; (4.) The absence of hemorrhages, other than occasional epistaxis; (5.) The constant absence of albumen in the urine; (6.) The difference in the anatomical lesions, and chiefly in the presence of enormously enlarged and softened spleens; (7.) The absence of all the lesions characteristic of *vomito*, such as the yellow color of the body, with marbled marks, black matters in the intestines, discoloring of the liver approaching to yellowness; (8.) Finally, the non-immunity of soldiers of the garrison who had already suffered from yellow fever (*Recueil de Mémoires de Médecine, de Chirurgie, et de Pharmacie Militaires*, No. 37, Janvier, 1863).

Considering true yellow fever, therefore, as one of the specific continued fevers, having a certain limited geographical range, it is necessary at the outset to define what is meant by true or pestilential yellow fever, and what are the diagnostic symptoms which distinguish it from the diseases which resemble it, but which are really dissimilar.

It may be asserted unconditionally at the outset, that the significance of the symptoms of yellowness of the skin and black vomit is very small indeed as diagnostic marks. Different shades of yellowness of the skin have been described as forming a prominent symptom, not only in epidemics of yellow fever, but by all writers on the fevers generally of hot countries. Yellowness of the skin in remittent fevers arising from malaria has been noticed in all climates, although it is certainly most common in those of the western hemisphere. Cleghorn observed it at Minorca; Irvine in Sicily, in the autumnal fevers; Burnett in the Mediterranean fevers, of all depths of colors. In the fatal fever of the Mysore country, yellowness during some years has been almost universal; so also in Batavia, and in the fevers of Rangoon in 1824-25. A fever attended with yellowness of the skin raged like a pestilence in Rohilcund from 1836 till 1840, at the same time that a fever with symptoms of plague was prevalent in Marwar and Meywar, and common remittents and intermittents prevailed between these districts. A fatal remittent fever attacked Her Majesty's 29th Regiment in 1844 at Ghazepore. In many cases there was "deep jaundice;" and in one case a symptom occurred which has been often witnessed in the West Indies—namely, sloughing of the penis and scrotum (Parkes "On the Contagion of Yellow Fever," in *Brit. and For. Med.-Chir. Review*, Jan., 1848). So also in some forms of specific yellow fever, as in the *algid* form, so well described by Dr. Lyons, in the Lisbon epidemic of 1857, yellowness was very often wanting, "many cases dying without having ever exhibited a trace of yellowness on any part of the cutaneous surface, or even the conjunctivæ during life" (Lyons *On Fever*, p. 338).

Black vomit is an event which occurs in fevers of marshy origin, and in the so-called "seasoning fevers," as well as in gastric affections of a purely tropical nature, in *coup de soleil*, and in some injuries of the brain. In the remittent fevers of the African stations, black vomit is not an unusual occurrence. So also in some of the

yellow fevers of America, which are of marshy origin, black vomit is a usual symptom (Boott).

These two events—namely, yellowness of the skin and black vomit being of themselves insufficient as diagnostic marks of true or specific yellow fever, additional grounds of difference are found,—(1.) In the type of the fever, which is continuous and not remittent; (2.) In the fact that it occurs, as a rule, only once during life; (3.) In the fact that it is propagated by specific media from infected persons or places to others. But although in no one of these phenomena, taken singly, except in that of its communicability from person to person, do we find any definite characters to rely upon to prove the existence of a formal and specific yellow fever, yet, in the general assemblage and collocation of symptoms, peculiarities do present themselves which are easily discernible by an experienced eye.

Though the subject of tropical fevers is too little known to warrant decided opinions on many points, yet the true yellow fever, or hæmagastic pestilence, is now so clearly stamped with characters so peculiarly its own, that it takes its place as a specific fever of a continuous and generally rapidly fatal type. Its pathology is best exemplified in the history of such isolated outbreaks of it as are to be found in the cases of the "Hussar" (BLANE), the "Bann," the "Kent," the "Scout," the "Eclair," the "Hankey," the "Icarus," the Lisbon epidemic of 1857, and the importation of the disease from Havana into the port of St. Nazaire by the "Anne Marie" in 1861; and no description of yellow fever can be complete which does not give an account of some of these remarkable instances of this disease.

An analysis of all the circumstances connected with the "Eclair" shows (1.) That the immediate consequences of landing the crew at Boà Vista were a thorough intercourse with the inhabitants, and the communication to them of the same fever with which the "Eclair" was infested. For some time before the arrival of the "Eclair" it is certain that the island of Boà Vista was perfectly healthy; and this was true also of all the other islands of the group (ALMEIDA, MACWILLIAM). So great, also, was the dread of the disease among the inhabitants that the consul had great difficulty in procuring laborers; nevertheless, the crew managed to smuggle vast quantities of spirits, and, of course, it is possible that more secret intercourse went on than can be gathered from any official reports. Certain of the inhabitants were also brought more or less in contact with the crew of the "Eclair." There were—1st. The military guard at the fort; 2d. The laborers employed on board the "Eclair"—forty-one in number; 3d. The laborers employed in the launches, or at a coal-heap on a small island—forty-six in number; 4th. Washerwomen who washed the officers' clothes—seventeen in number. In addition to these, Captain Estcourt, the commander of the steamer, lived at the consul's house; the gun-room and ward-room officers and midshipmen occupied a house in Porto Sal Rey; and leave was given to the warrant officers and a few of the men, one of whom stopped in the town for two nights. The

"Eclair" left on the 13th of September; on the 14th or 17th a corporal of the guard was taken ill; on the 15th or 18th, a private; and in both cases the symptoms were fever, wildness, and constant black vomiting. The corporal died on the 17th or 20th, and the private on the following day. Others of the guard were taken ill, and, being conveyed into the town, the introduction of the fever amongst the inhabitants is attributed to them. It also appears (2.) That the men who were chiefly in contact with the crew and with the sick men, and who were in the sick men's apartments, suffered much more severely than any other class; (3.) That the propagation of the fever appears to have been strictly in proportion to the amount of intercourse; (4.) That within a reasonable time after the departure of the "Eclair" there were three persons ill with fever at Boà Vista, and two already dead—all of whom had been in contact with the crew of the steamer.

The period of incubation of the fever at Boà Vista was found to vary from two to eight days; and the facts recorded in the history of the spread of the fever over the island show that certain persons living nearest and most in contact with the two sick soldiers were first attacked. When the disease appeared with great virulence in the island of Grenada in 1793, its spread by infection first attracted notice by the arrival of the "Hankey" from Bulam, on the west coast of Africa, on the 19th February, 1793, some days before the fever broke out on the island. In this vessel, at Bulam, the fever had prevailed for five months before, to a great and fatal extent (CHISHOLM, SIR WM. PYM).

A most interesting and consistent account of a yellow fever outbreak has been given by Surgeon J. D. Macdonald, Esq., F.R.S., in the medical journal of the "Icarus" (*Annals of Military and Naval Surgery*, p. 126). Mr. Macdonald's account is especially interesting in the following particulars: (1.) It tends to fix more definitely than has yet been done a period of incubation. (2.) It shows that in certain localities specific yellow fever is always in existence, expressing itself by sporadic cases every now and then, especially in the West India Islands, as a central focus to that geographical district, where the disease is endemic, and where it has assumed in some places an epidemic character. (3.) It tends to corroborate the circumstantial evidence already accumulating, that the disease may be propagated by *fomites*, much as in the case with typhus and scarlet fevers. (4.) As with typhus and typhoid fevers, so with yellow fever, "the doctrine of its spontaneous origin" can have no foundation to satisfy the rational mind.

Bearing on the *period of latency*, Mr. Macdonald shows that yellow fever was rife at St. Domingo when the "Icarus" arrived there, and the professional zeal of Dr. MacLagan, the assistant surgeon of that ship, led him to visit some half-dozen cases on shore at Port-au-Prince. He is believed to have visited these cases on the 10th of June, and the first appearance of the symptoms which ended in his much-lamented death betrayed themselves on the 24th of June—*i. e.*, fourteen days after having first visited the sick, and after the ship had been at sea four days. Fourteen days is not uncommon as the

period of latency for the variolous poison. Another case is cited—that of a boy, Lambert—which shows that the period of latency could not have been less than *eight* days. The experience of the Lisbon epidemic marks the time of incubation as varying from two to ten days, and in some instances extending to fifteen days. The importation of yellow fever by the ship “Anne Marie” into St. Nazaire—a town in the department of the Loire, about 47° 30' north latitude—in the summer of 1861 (*Ann. d'Hygiene*, Oct., 1863, p. 416), confirms the belief in a lengthened period of incubation; and from a careful analysis of the history of specific yellow fever cases, I think it will be found that the period of incubation tends to lengthen with the transportation and propagation of the disease into latitudes the most remote from the equator.

The history of the importation of the disease into St. Nazaire is as follows: About the 13th of June the “Anne Marie,” a wooden sailing vessel, laden with cases of sugar, left Havana, having been there a month during the prevalence of a severe epidemic of yellow fever. None of the sailors suffered so long as she lay at Havana, except from a little depression, loss of appetite, and a certain tendency to vomiting. After leaving Havana for France there was no sickness for seventeen days. On the 1st of July two sailors were attacked (without precursory symptoms) with violent shivering, pallor of the face, injection of the eyes, congested lips, and continued delirium. One died in twenty-three hours, the other in one hundred and ten. On the following seven days other persons were attacked, making in all nine cases out of a crew of sixteen persons. Only two deaths occurred. The ship arrived at St. Nazaire on the 25th of July with seven men still sick, but all of them convalescent, thirteen days having elapsed from the date of attack of the case last taken ill. Therefore, having had no deaths and no fresh cases for ten days, the “Anne Marie” was not placed in quarantine at the end of her voyage on arrival at St. Nazaire. Near her, as she lay in that port, there were anchored two ships of the imperial navy—namely, “Le Chastan” and “Le Cormorant,” the former touching her. Three other ships, “L'Orient,” “Les Dardanelles,” and “L'Arequipa,” were also near her. According to the custom of the port, the sailors of the “Anne Marie,” being only engaged for the voyage, quitted the vessel on her arrival, and were dispersed throughout the town. The commander, who had been ill, went home, the vessel was left to the second in command, and the places of the men were taken by seventeen fresh men, to discharge the cargo of sugar. These men were strong, very robust, and completed the discharge of the ship in eight days. Of these men twelve or thirteen were attacked with yellow fever, and many of them died.

“Le Chastan,” having been close alongside the “Anne Marie,” left on the 29th July, and sailed to Indret, on the Loire, 44 kilometres distant. Her crew, five in number, seemed in perfect health when they arrived at Indret; but on the 1st of August (*i. e.*, three days after leaving St. Nazaire) a man fell sick of yellow fever; then the remaining four by the 5th of August were all ill; and by the 10th of August all the five men, the crew of “Le Chastan,” were

dead. These men had all been on board the "Anne Marie" for about a quarter of an hour. "Le Cormorant" quitted St. Nazaire on the 10th: her crew, six in number, were then in perfect health. On the 14th of August two of her men were taken ill, and died on the 26th, of decided yellow fever.

Eight vessels in all had been near the "Anne Marie," and sick patients had gone on shore at St. Nazaire and its neighborhood, communicating yellow fever to two or three, and slighter illness of the same kind to other persons who were about them. Altogether, it is on record that forty-four cases of yellow fever were communicated by the "Anne Marie," resulting in twenty-six deaths. During this period the heat was more like that of a tropical than of a European climate; but neither yellow fever nor anything like it had ever been seen in the district. It cannot now, therefore, be disputed that, by the arrival of the "Anne Marie," yellow fever was imported into St. Nazaire, and by propagating itself occasioned an outbreak of yellow fever at that port. And when it is remembered that fatal cases of specific yellow fever have already occurred at our own doors,—in Plymouth Sound and Southampton Water,—there is a like possibility of the importation of yellow fever into English ports under climatic conditions favorable to its development, and when we might least expect it, through the rapid and frequent communication which we now enjoy with the West India Islands, the very central focus of yellow fever. "We have no more reason to trust in our own fancied security than the people of Monte Video had before yellow fever made its appearance amongst them" (J. D. MACDONALD). The *Lancet*, of Feb. 12, 1863, contains "An Account of Yellow Fever as it occurred on board the Royal Mail Steamship 'La Plata,' in the month of November, 1852," from the pen of my friend Dr. Wiblin, the medical superintendent of quarantine at the port of Southampton. He there shows that fourteen cases of yellow fever had occurred on board the "La Plata" during her homeward voyage to Southampton from the West Indies. On the morning of the 18th November she arrived at Southampton, where she was kept for two days in quarantine. On the morning of the 28th the fourth engineer of the "La Plata" (Mr. Napier), who had been lodging in the town for eight days, was seized with symptoms of yellow fever, and, after a week's illness, died of the disease. No other cases occurred in the town. The "La Plata" was a wooden ship; and Mr. Napier had been at work on board of her during the time when he was lodging in Southampton.

In connection with the extension or propagation of specific yellow fever beyond its usual geographical limits of constant existence, "it is a remarkable fact," says Mr. Macdonald, "that the deaths from yellow fever at St. Thomas's, before it became a coaling depôt for the Royal Mail steamers, as compared with the deaths afterwards, bear the proportion of 4 to 64. And in connection with this, must also be mentioned an equally important fact, that the combustible and other qualities of coal, exposed to the weather as it is at that island, became much deteriorated." How far this loss may prove to be a *pabulum* to yellow fever poison is unknown, but the cover-

ing in, as well as the inclosure of coal in tropical climates, as Mr. Macdonald notices, is worthy of the attention of the proper authorities.

All the cases, like the "Eclair," the "Bann," the "Imaum," the "Icarus," the "Anne Marie," the "La Plata," agreeing as they do in all their main features, it is impossible to doubt the existence of a multiplying infecting virus as the specific cause of yellow fever; and in cases where it was transported, imported, and propagated, the fever, except in certain cases, seems to have arisen only in persons who had been exposed to whatever deleterious influence was exercised by the atmosphere of ships in which cases of yellow fever existed. Moreover, the facts connected with the "Imaum," the "Icarus," and the "Barracouta," show that infected places and persons are alike dangerous to those who are at all susceptible; and in the cases of the "Imaum" and the "Anne Marie," it was shown that a disease taken in a certain locality, and spreading from person to person, may finally affect a second locality through their medium. It appears also to be quite a mistake to suppose, as Mr. Macdonald observes, that no individual can communicate the disease to another unless he himself is actually under its influence at the time; or, secondly, to consider such an individual as differing in any essential particular from an infected locality. Indeed, a ship itself is only an individual on a grander scale. This is the view Mr. Macdonald takes with regard to the "Imaum," receiving the disease from the officers of the "Icarus;" and it is also the view which explains the events which followed the contiguity of the ships to the "Anne Marie" in the harbor of St. Nazaire. To some extent the disease spread through personal intercourse to persons who were not near the ship,—in one very important case, that of M. Chaillon, a physician at Indret, who is said never to have been near the ship nor the town of St. Nazaire, but who contracted infection from four laborers who came infected from the ship, and whom he attended medically at their houses. He contracted yellow fever, and died. In a second case, one of the ship laborers, who himself had yellow fever, is said to have carried the infection certainly to his wife, and perhaps to an old man in whose house he and his wife lodged. All of them were attacked with yellow fever—the old man fatally. Unquestionably with regard to the "Anne Marie," and doubtless also with regard to the other ships, *the ships themselves*, irrespectively of sick persons in them, were *foci* of yellow fever infection. The men therefore, no doubt, carried infection passively, as they might have carried an odor from the ship, or as a student carries the smell of the dissecting-room on his clothes, especially felted textures. "I have often looked upon my own monkey-jacket with horror," writes Mr. Macdonald, "as the possible means of communicating so formidable a disease to others." Men thus laboring in the hold of infected ships, without themselves contracting yellow fever there, might carry infection to their homes, in climates and places where yellow fever may prevail. And it is a question how far, like cholera and typhoid fever *excreta*, the poison of yellow fever may not increase in places where decomposing animal material abounds.

With regard to the Lisbon epidemic, it has been proved that the epidemic remained concentrated in Lisbon, and did not extend to its neighborhood, nor to any other part of the kingdom, although the communication by land with all the towns and villages continued active and uninterrupted. In many places there appeared cases of yellow fever, evidently brought from the capital, but in no place was the disease transmitted or propagated in an epidemic form. The official report records, that of 182 persons who left Lisbon for different places in Portugal, carrying with them the germ of yellow fever, which broke out or developed itself in them after their arrival at those places, 86 of them died. *In no instance was yellow fever communicated from them to any other person in the places whither they went.*

As to the origin of the disease in Lisbon, it appears certain that the first cases were amongst men employed in the custom-house, or with persons in close communication with them. It showed itself in the streets where these people lived. The disease, which commenced in July, did not become epidemic till September; but it remained stationary for awhile in the parishes where it first broke out, and then spread gradually and regularly to other parts. It is therefore presumed that the disease did not proceed from any general cause operating on the whole mass of the population, such as would come from meteorological or hygienic conditions. It was not persons of different classes, and living in different parts of the city, who were attacked at the same time; on the contrary, it commenced with one distinct class, between the individuals of which there was much communication, and went from house to house, from street to street, without invading more than a certain extent of the city. It is presumed, but not proven, that the disease was imported through the custom-house, where luggage and effects are opened to be passed. Many of the ships which arrived at Lisbon during the epidemic and in the previous months had cases of yellow fever and losses during the voyage; but the only case proved was in the "Tamar," which arrived in March from Brazil, the captain of which owned that he had two deaths from yellow fever on board during the passage. This ship arrived again in September, and reported at Southampton two cases which she had had on board. Lisbon and Oporto have been very subject to these importations since 1849, when yellow fever became epidemic in Brazil. In the report it is concluded,—(1.) That Lisbon is not to be considered as liable to spontaneous outbreaks of yellow fever. (2.) But, from its latitude and local condition (especially inasmuch as it abounds in filth in some localities where population is dense and sewerage imperfect), it is favorable for the development and spread of specific yellow fever. (3.) It is concluded that the epidemic of 1857 was imported from Brazil.

[The following facts, favorable to the doctrine of the transmissibility and portability of yellow fever, are mainly condensed from the valuable *Report on Epidemic Cholera and Yellow Fever in the Army of the United States, during the year 1867*, by Dr. J. J. WOODWARD, U. S. A. (Circular No. 1, War Department, Surgeon-General's Office, Washington, June 1868.)

In June 1862, yellow fever broke out at Key West, Florida, having been

imported from Havana by the bark *Adventure*, which put into Key West in distress, about the 20th June. It lay in quarantine thirteen days. Sixteen days after leaving Havana, the first and second mates fell ill with the disease, and in two days these men and two others of the crew, suffering from the fever, were taken on shore and placed in the Marine Hospital. July 27th, a soldier of the 90th New York was attacked with yellow fever, which subsequently spread through the garrison, there being 331 cases and 71 deaths.

The steamer *Delaware* with a detachment of the 7th New Hampshire, arrived at Hilton Head, S. C., from Key West, early in September, 1862, and, after a short quarantine, landed her passengers September 8th. Soon after several of these were taken ill with the fever, and eight died. On the 9th October a quartermaster's employee, living close by the quartermaster's depot in which a lot of tents brought by the *Delaware* had been stored, was attacked; subsequently, a number of officers, soldiers, and men, employed in the quartermaster's department, and all living around the storehouse in which had been put the tents brought from Key West by the *Delaware*, became affected. The hygienic conditions around this wharf were bad. The number of cases of the second outbreak was 30 and 17 deaths. The disease did not spread amongst the troops in garrison, nor did any of the physicians, attendants, or patients, suffering from other diseases in the general hospital, where the cases of yellow fever were treated without separation, contract the disorder.

In the same year (1862) there were outbreaks of the fever at Charleston, S. C., and at Wilmington, N. C.; and there is good reason to believe that it was introduced into both ports by blockade-runners from Nassau, N. P., where it prevailed.

Early in the autumn of 1864 yellow fever appeared at Newbern, N. C., and continued until the end of November; 705 cases and 288 deaths were reported amongst the white troops, and 38 cases and 15 deaths among the colored troops. Evidence is wanting to show the exotic origin of the disease, and it is claimed that it was of domestic generation, the local hygienic conditions being excessively bad. Previous to the outbreak at Newbern, the fever had appeared at Charleston, S. C., but there is no proof of any communication having been had between the two towns.

On the 10th October, 1864, the first case of yellow fever appeared at Wilmington, N. C. About the last of the previous August, two blockade-runners, with cases of the disease on board, were at quarantine three miles below the town. The quarantine was subsequently removed to near the mouth of Cape Fear River, and here, on October 1st, there were fourteen blockade-runners, and on all of them the mortality from yellow fever was very great. At this time the fever spread from the ships to the shore, the first cases being in the houses nearest the quarantine, and nearly one half of the inhabitants of the town died of it. Goods were known to have been smuggled on shore, and it is very likely some of them were carried to Wilmington.

During the autumn of 1864, 191 cases and 57 deaths occurred on board twenty-five naval vessels lying in the Mississippi River about and below New Orleans. It is claimed that these cases were spontaneously generated, and the bad hygienic condition of the iron-clads furnished all the alleged necessary factors. But the Spanish man-of-war *Pizarro*, with yellow fever on board, had been sent on the previous 4th of July to the quarantine, the first cases of the disease appearing on the 12th September. There were 12 cases and 3 deaths among the employees and guard at the Naval Hospital and the boat-landing at Erato Street; and five cases of

black vomit happened among citizens exposed to the same cause in the vicinity of the landing.

In the summer of 1867 there were outbreaks of yellow fever at Indianola and Galveston, Texas, and at New Orleans. Dr. Woodward says: "The more thoroughly the facts connected with the spread of yellow fever in the army during 1867 are known, the more strongly they appear to favor the theory of the exotic origin of epidemic yellow fever in the United States" (*loc. cit.*, p. xviii).

The reports indicate clearly two foreign sources from which the disease was imported into the United States last year,—Vera Cruz, and Havana. From Mexico it was taken to Indianola, and thence carried to other points in Florida. At all other stations it seems to have been carried directly or indirectly from Havana. The cases of Mexican origin were more fatal than those of Cuban origin, 2 out of every 5 cases of the former dying, and but 2 out of 7 of the latter. From Indianola the pestilence was carried to Galveston, and from each of these points it spread towards the interior of the State, along the chief routes of travel.

The fever was introduced into New Orleans from Havana. It spread from New Orleans to Ship Island, to Baton Rouge, and to all other places where it afterwards appeared in Mississippi, Alabama, and Tennessee. At Key West, and at Fort Jefferson, Tortugas, it was brought directly from Havana.]

Symptoms.—Uniformity in the order and character of the symptoms of specific yellow fever must not be looked for. All the best writers on the subject, whether recording their experience in the West Indies, the west coast of Africa, or the south coast of Spain, are unanimous to the contrary, and consequently, while direct and faithful descriptions may have been given of each epidemic, yet the results are not general nor uniform. Certain symptoms in certain epidemics vary in their nature and in the time of their accession, while others common to former visitations are wanting in those which follow. Sometimes the full complement of standard symptoms are present, sometimes they are imperfect and deficient, and sometimes displaced. At one time the diagnostic symptom in an epidemic is the supra-orbital headache. At other times the tongue symptoms are alone diagnostic, or their equivalents are expressed in the uvula and fauces. Intense surface heat, albumen early in the urine, and early black vomit, are among the later symptoms; and smoky pale urine, with perfect blood-corpuscles, take the place of the straw-colored or bilious urine, with its sediment of tube-casts and epithelial matter.

The general appearance of the tongue in well-marked cases is redness of the tip and edges, with prominent papillæ and a creamy surface. Subsequently the fur separates from its middle surface, and lies in white, wavy flakes; and the next series of changes consists in separation of the epithelium, which begins at the tip, proceeds to the edges and down the raphe, and may continue till the whole surface is denuded, the papillæ obliterated, and the tongue becomes smooth and dryish, of the color and appearance of raw beef. Such a condition is generally associated with exudation of blood so free as to coat the mouth and tongue, and collect upon the teeth. The tongue is then usually much smaller and more pointed than in the earlier stages.

A rare manifestation of capillary irritation in yellow fever, consists in an efflorescence of the skin in the form of a subcutaneous rash on the chest, and extending over the abdomen and arms. Rose-colored spots, of a somewhat circular shape, have been noticed on fine, delicate, sensitive skins, varying from the size of a flea-bite to what might be covered with the point of the finger. They result generally from mosquito-wounds, and become hemorrhagic at the end of the disease when it terminates fatally.

Bloody furuncles appear late in the order of symptoms, and are to be regarded rather as sequelæ. Their most common site is on the wrist, over the metacarpal joints, along the front of the legs, below the scapulæ, and over the hip, in the parotid region, and over the forehead and lip. They are generally in close proximity to the smaller arterial branches, such as the ulnar, radial, anterior, tibial, gluteal, intercostal, and facial arteries. They become tender, acuminated, and inflamed, and sometimes form large abscesses of purulent matter, with a pale or inflamed surface, and this chiefly when below the scapula or over the hip. Generally on the legs they are flat, present no inflamed appearance, but show a flat purplish vesication, about the size of a split pea or a sixpence.

Two, three, or even four forms or types of yellow fever have been described by authors. These have been very clearly defined by my friend, Dr. Lyons, of Dublin, in the Lisbon epidemic of 1857, which he investigated with so much care and enthusiasm.

The *types, groups, or forms* which he found capable of clinical recognition are,—(1.) The algid form; (2.) The sthenic form; (3.) The hemorrhagic form; (4.) The purpuric form; (5.) The typhous form.

The first of these, namely, the algid form, is that which presents the most rapid course, and the earliest and greatest amount of prostration of the vital powers. These are the cases which are suddenly killed with the poison. "The patient, while in the enjoyment of his usual health, and in the midst of his usual occupation, feels suddenly the effects, as it were, of a sudden blow from a heavy bar on the back, falls down while walking (or if standing), and dies within a few hours in profound collapse, and after exhibiting more or less of the other symptoms of this fever." The countenance became sunken, the eye dull and filmy, the surface cold, and the patient felt cold, depressed, and wretched. The face became of a dirty livid hue, and this appearance extended to the trunk and limbs, the surface then presenting innumerable points of minute venous congestion, and sometimes purpuric spots and patches of various sizes. In extreme cases the lips, the breath, and tongue were cold, with a temperature in the axilla not more than 96°, the pulse being small, feeble, and quick; and when the cardiac action became feeble the radial pulse would be obliterated.

The *sthenic form* is a marked contrast to the algid. It is especially well marked in both sexes at the prime of life, and in persons with well-developed muscular frames. Such cases are characterized by well-marked febrile symptoms, severe and persistent headache, much rachialgia at the outset, a high, full, and hard pulse, occasionally thrilling and resisting, with flushed face and throbbing

temples. A remarkable elevation of temperature prevailed—an increase of 3° , 4° , or even 5° Fahrenheit, and in some an increase of 7° Fahrenheit was observed. Death sometimes took place in a very unexpected manner.

In the *hemorrhagic type* the cases are the most characteristic and appalling. In them epigastric anxiety, with or without heat, and pain on pressure in the epigastrium, is well marked; but their great characteristic is a tendency to profuse simultaneous effusions of blood from various parts and organs—the *hemorrhage never being single, nor from any one source or organ only*. The cases are fatal at an early period; and all the connective tissue of the body is surcharged with blood. There is less considerable elevation of temperature than in the sthenic form.

In the *purpuric form* the pyrexial state is well marked, with the conjunctivæ and general surface intensely yellow. Purpuric patches commence and spread—sometimes with surrounding œdema. These patches are manifestly caused by subcutaneous effusions of the coloring matter of the blood; and all varieties and shades of colors and tints are observable.

In the *typhous form* two orders of phenomena may prevail. In one class of cases stupor and nervous depression exist from an early period of the fever, with all the other well-marked and characteristic *typhoid* symptoms; and to these are superadded the hemorrhagic phenomena. In another class of these cases the patient, after passing through the sthenic or the algid form, would insensibly glide into the typhoid state, on the cessation of the hemorrhages (LYONS).

It is of great importance to attend specially to the study of these forms or types; for, as Dr. Lyons justly observes, much of the discrepancy and apparent conflict of medical testimony on the subject of yellow fever is due to the want of discrimination of those leading characteristics and salient features (LYONS, *l. c.*, p. 375).

When the black vomit is plentiful, or the urine free, the intelligence remains clear and unclouded, but the skin becomes cold and damp, the pulse small, and finally extinct at the wrist, and the patient dies of gradual *exhaustion* and *syncope*.

According to the amount of febrile excitement, the skin is hot and dry, but the experience of Mr. Macdonald leads him to say that in many cases there is a turgid fulness of the vessels, and a tingling heat of the surface, which is imparted in a remarkable manner to the fingers on feeling the pulse. This feverish heat of skin Mr. Macdonald thinks is much more moderate in cases treated in the open air than in those treated in the wards of an hospital, however well ventilated.

There is great irregularity in the temperature of the surface. Sometimes the forehead is the hottest part of the body, occasionally it is the chest. The uncovered parts, in the latter stages of the disease, are easily reduced in temperature, and thus, while the exposed chest and extremities may feel cool to the touch, the axilla may raise the thermometer to 102° or 103° Fahr. The highest temperature Dr. Blair has observed was 107° in the axilla.

Mr. Macdonald notices that the pulse is at first quick, and of

considerable strength, though still compressible, and may vary in these respects within certain limits until it becomes feeble. In those cases which have proved rapidly fatal there has been a marked gradual decrease in its strength; and finally, when the ferrety eye grows clear, and a pallor of countenance shows the mischief of blood exudation going on within, it is scarcely to be felt.

Observations made on the urine, in yellow fever, by Dr. Blair, show that it is always acid in the first stage, and continues so generally till convalescence, when it becomes alkaline, or until it becomes heavily charged with bile. During the early stage the urine is normal in color, clearness, and quantity. About the third day the color alters, and becomes of a sulphur, primrose, straw or light gamboge hue, perhaps slightly turbid, and with a little floating sediment. The color deepens till it becomes yellow or orange; and if the case ends in convalescence, the urine is copious, and may appear black. Sometimes the urine has a pale, watery, smoky appearance, with a layer of blood-corpuscles in the sediment, and sometimes it is very bloody.

Albumen appears on the second or third day generally; in some cases as early as the first day; and in a few cases not till the day of death, and after black vomit has set in. Albumen appeared in every fatal case of normal duration. It sometimes ceased suddenly in convalescence, and always before the yellow suffusion of skin and eye, or bile in the urine, had disappeared. Between the eleventh and twentieth day of grave cases it generally disappeared. Its color was never white. When the urine appeared turbid, it was due to the presence of mucous epithelial matter, coagulated albumen, coats of the urinary tubuli, or fine capillaries of the kidney or mucous membrane passed out with the urine. The tube-casts are generally short, thick, club-shaped, and opaque, attended with large organic cells and epithelial scales. Crystalline deposits are rare. In females the catamenia are sure to appear, whether due or not. No sign is so dooming as a suppression of urine, *black vomit* not excepted. The alvine evacuations may be black towards the close of the disease, or very dark green, and bilious; but after the black stools have ceased, they are succeeded by evacuations which resemble fine, dark, sandy mud, and named the "caddy stool." As the disease still further advanced, and towards its fatal termination, the dejections again changed their character. They became scanty and mucous, of various consistence and color. These mucous stools almost always appeared *after* black vomit, and were contemporaneous with the scanty urine before described. The alvine evacuations in yellow fever, from the beginning to the end of the attack, are always alkaline, except in one instance, that of the black vomit stool; in that it is always acid. Its chemical quality is evidently due to the admixture of a portion of the black vomit, which has descended (if not found in the intestines) by peristaltic motion into the intestines, and mixed with the scanty mucous stool, and in such quantity as not only to neutralize it, but to be in excess. The scanty thick mucous stool—almost a jelly—has generally a little thin serum around it in the bottom of the pot. The bulk of all these varieties of the scanty

mucous stool consists of mucus, broken-up epithelial matter, and myriads of epithelial granules. Sometimes little wavy flakes, like morsels of cuticle, are also to be found. They also frequently contain the crystalline bodies of the caddy stool, particularly when they are rather thin and serous. By appearance, they would be taken for rectal stools, and the results of tenesmus; but such is not the case. A burning sensation is often complained of, but seldom any tenesmus, and no doubt these stools consist of that mucous matter which we find after death lining the intestinal canal generally. In a few cases where there has been total suppression of urine, these stools have become diarrhœal.

The first ejections from the stomach of a yellow fever patient are seldom seen by the physician. Mucus and bile soon appear, occasionally with a streak or speck of blood, and with violent retching. The ejections are alkaline. Generally after the first vomiting the stomach becomes tolerably settled, until the second stage sets in, on the second, third, or fourth, or as late as the fifth day of the disease. Then, without warning or nausea, but on any trifling provocative, the stomach suddenly ejects a quantity of clear, pale, limpid, or slightly opalescent *acid fluid*—the *white vomit*, which indicates the beginning of the stage of acid elimination, and is generally contemporaneous with the first shedding of epithelium from the tongue. Sometimes the evacuation of this vomit has a critical effect, equivalent to the perspiration of intermittent fever. True white vomit consists of serum, more or less acid, which remains clear on the application of heat and nitric acid.

The transition of symptoms from *white to black vomit* is generally gradual; and is attended with a "suspicious sediment" of "snuff-like specks" before it merges into well-defined *black vomit*. The stage of acid elimination continues to the close of the disease, and is most intensely manifested during the production of the *black vomit*. The presence of ammonia in black vomit is universal, and may be considered as one of its tests; and its specific gravity 1.004 to 1.006, the temperature of the air being 86°. Its sediment consists of coagulated albumen and the debris of blood-cells. Another test is acidity, and a third is to be observed in the phenomenon that the sediment is dissolved by liquor potassæ, which disengages ammonia.

Another feature in the pathological symptoms of yellow fever may be expressed by the fact that the urea of the suppressed urine is eliminated from the system as a volatile salt, by metamorphosis into a carbonate of ammonia, which as such is frequently found in the breath, in the normal black vomit, in combination with an acid, almost always in the stool, and apparently pervading all the tissues of the body. "It would seem," writes Mr. Macdonald, "as though the mucous membrane of the stomach were called upon to compensate for the defective secreting and eliminating power of the kidneys; and in those cases where little or none of the matter of black vomit was found in the stomach after death, the lining membrane presented a thickened appearance, with a muco-sanguineous coating" (Grant's *Annals of Military and Naval Surgery*, p. 135).

Prognosis.—The data to judge from may be arranged into symptoms which are favorable, and those which are not so. (1.) *The favorable symptoms are*—A slow pulse and moderate temperature of the body, and quiet stomach. Streaks of blood during the stage of black vomit, or after acid elimination has set in, are favorable, if the corpuscles are found entire. If the urinary secretion continue, and the black vomit be scanty from the first, or is afterwards suppressed, the patient may yet survive. Urine simply albuminous is a less serious sign than when it also contains tube-casts. Free, copious urine, no matter how dark or bilious, is the most favorable of any single sign. Prognostics may be derived from the effects of treatment. (2.) *The unfavorable signs are*—The more fiery crimson the tip and edge of the tongue, the more irritable the stomach, the severer the headache, the worse the prognosis of the first stage, and *vice versâ*; but a streak of blood in the early vomit indicates much danger from the attack. In the second stage the earlier or more complete the suppression of urine, and the more copious the ejections of black vomit, the more imminent the danger. If the urine be scanty, and loaded with tube-casts, entangled in epithelial and coagulable matter, the light buff-colored curdy sediment before mentioned indicates a complex lesion of the secreting structure of the kidney. It is the urine symptom in its maximum of severity, and is as fatal as if the suppression had already occurred. Blood-corpuscles in the urine are not to be looked on with apprehension. A faltering of the articulation is a bad prognostic, and a difficulty of protruding the tongue enhances it. The danger of the case is enhanced by inflammatory complications, and by hypertrophy of the heart. A recent residence in a temperate climate; the race or complexion of the individual; the fact of his previously having suffered from an attack, will enter into an estimate of his chances of recovery.

Prognosis is declared by Robert Jackson to be treacherous and difficult in the extreme.

Treatment.—An early attention to first symptoms among the susceptible is of the greatest value in saving human life.

The diagnosis of cases in which the attack has been said to have been “aborted” by remedies may be questioned, and such a belief is opposed to the doctrines of sound pathology. Moreover, the “heroic” doses of calomel which were given in such cases, combined with quinine, cannot be too strongly discountenanced, for “they were first recommended on the strength of a crazy hypothesis” alone. The practice is said to have frequently proved successful in Jamaica; but, according to Dr. Davy, it was not attended with beneficial results at Barbadoes; and the American physicians at New Orleans have not found it to answer their expectations in stopping the fever.

The large and frequently repeated doses of quinine were often also highly injurious (LAWSON). This discrepancy may in some measure be explained by what has been stated at the outset in explaining the pathology of this peculiar fever. It is in cases where the fever is of the periodic or paludal form, and not the continuous or true yellow fever, that quinine may be of use, if the system

can be brought under its influence. The ill effects of quinine in specific yellow fever consist in its checking secretion and deranging the circulation within the head; and Mr. Macdonald considers its employment more suited to the convalescent than to the patient immediately under the influence of the disease.

It is an object to keep the bowels freely open, and to get the skin to act freely. The main object of the physician should be to moderate excessive action in any organ, and to endeavor to bring about as complete a crisis as possible about the fifth day, which seems to be the natural period of resolution of the disease. For this purpose nothing is of more importance than to re-establish the secreting function of the colon, and to obtain feculent evacuations—not mere bilious discharges—but proper dark-brown feculent stools (LAWSON). Gentle excitement of an extensive portion of the lining membrane of the colon, with frequent copious enemata of a pint and a half of warm water, in which a tablespoonful of common salt has been dissolved, and to which has been added a tablespoonful of olive oil, or more stimulating enemata, such as of turpentine, deserve a full and careful trial. All the depurative functions must be kept in activity. Turpentine is recommended by Dr. Copland, by Dr. Archibald Smith, and Mr. Laird, of H. M. S., “Medea”—one drachm doses by the mouth, or half ounce doses as a lavement, several times daily, using it also as an epithem on the abdomen.

“When the mucous surfaces,” writes Dr. Blair, “as indicated by the tongue, were denuded of epithelium, the use of *gum water* was decidedly beneficial. It lubricated, defended, and soothed the raw surfaces. The strength was generally three drachms of the purest powdered gum arabic, dissolved in six ounces of cold water, and a tablespoonful of this given every one or two hours. The patient at last gets tired of it; but for thirty-six or forty-eight hours of the most critical period of the disease it is used without dissatisfaction, and then can be substituted by, or alternated with, smoothly and thin-made arrow-root. When the heat of surface was ardent, a *wet sheet* or *blanket* was used for the reduction of temperature by evaporation, with frequently very good effect; (and if ice could be obtained, its internal use is well worthy of a trial). But in the latter stages of the disease, when the skin was cool or cold, the patient seemed to have an instinctive craving for its re-application, and frequently asked to be put into it. There would appear to be two causes for this feeling. We find it to exist in cases in which black vomit has been copious, and the associating thirst distressing; also in cases where there has been no black vomit of any consequence, and the breath is highly ammoniacal. In the former class of cases the stomach ceases to be an *absorbing* viscus in anything like the proportion of its secretions and transudations. The skin is therefore employed in reducing the crisis of the blood by the absorption of water, as shipwrecked mariners are said to quench their thirst. But not only does the skin afford an inlet for the imbibition of diluting fluids, but the softening of the cuticle would seem to afford an additional outlet for the noxious elements of the circulation; and it is probably in this direction we must in future look for

auxiliary means of relieving the blood of its poisonous, metamorphosed, and effete constituents, the onus of which is now thrown on such vital organs as the stomach and lungs. At one time the heat of the surface was so ardent and persistent that the wet sheet failed to reduce it effectually."

The most distressing symptom in yellow fever, both to the patient and the medical attendant, is irritability of the stomach; it is so constantly present, and so often uncontrollable, that the knowledge of every available means of checking it is of the utmost importance.

The food during the course of yellow fever should be of the blandest description—chicken tea, arrow-root, sago, and barley-water constituting the chief articles; and these should be taken in minute quantities at a time when the stomach is at all irritable. This rule applies to drinks of all kinds. The patient is greedy for a large draught of fluids; but by sucking them through a glass tube, or a straw, or a hollow reed of small bore, or by the tea- or tablespoonful, they are much more likely to be retained. A cold infusion of oatmeal was found an agreeable drink for Scotch seamen, of which they did not seem to tire. A dislike of sweets was observed among the patients; and when lemonade was asked for, the usual quantity of sugar was objected to, probably from its rendering the liquid too dense for ready absorption by the stomach, and therefore less quenching. Tea was found so uniformly to disagree with the patients, and cause vomiting, particularly in the advanced stages, that at length it had to be expunged from the yellow fever dietary. Dilute alcoholic drinks were given freely, and with good effect. Where brandy could be obtained pure (tolerably free from acidity and fusel oil), and was well diluted with water, that spirit answered every indication. Sometimes the effervescing wines were relished and retained, but they are very liable to the objections of containing foreign matters and the products of mismanaged fermentation (BLAIR).

Mr. John Denis Macdonald has several times proved that four or five minims of chloroform prepares the stomach for the reception and retention of food, by lessening its irritability, and the dose should be repeated a short time before food is again taken, as the effect of the chloroform is transitory. On the same principle the administration of *chlorodyne** may be advocated. Lime-water has been also found

* The composition of *chlorodyne* is variously given by chemists, but the following formula may be given as a very useful one:

R. Chloroform, fʒiv; Æth. Sulph., fʒii; Theriacæ, fʒi; Mucilag. Acaciæ, fʒi; Morph. Muriat., gr. viii; Acid. Hydrocyanic dil. (2 per cent.), fʒii; Ol. Menth. pip., ℥iv, ad vi; *misce bene*.

Syrup or water may be added to the mucilage of gum, and tincture of cannabis Indica (5 to 20 minims), or other anodynes, may be added at the time of prescribing, if deemed desirable. The difficulty in compounding *chlorodyne* is in getting the chloroform to mix with the treacle. It will not do so alone, but the use of a little thin gum, or even water, effects their mixture almost at once. The morphia ought first to be dissolved in the chloroform; then mix with the treacle the gum or water, first using about the same bulk of chloroform and treacle, afterwards adding the rest of the chloroform by degrees, constantly shaking briskly the bottle in which it is made up;

to have a most beneficial effect in allaying vomiting, and thus enabling the patient to partake of food; and the essence of beef is well adapted for such cases.

During the course of the disease, *auxiliary treatment* may be required to meet contingent symptoms. This is embraced chiefly in the use of sinapisms and stimulating liniments. Tenderness over the liver may be benefited by them.

Dr. Blair writes that when the primary reaction was violent, and the face was turgid, and the head symptoms severe, arteriotomy was performed, and with benefit. In a few such cases, and when the patient was young, strong, and full-blooded, and where the dynamic congestions were so violent that the vessels yielded to the turgescence and impulse, and blood-corpuscles without tube-casts, or even but a haze of albumen, was present in the urine, a vein in the arm was opened, and free bleeding relieved the tension of the vascular system. In such cases convalescence was slow and unsatisfactory, but the immediate results were beneficial. The severe pain in the loins, which is a constant symptom in yellow fever, is generally associated with renal congestion; and sinapisms or stimulating liniments afford great relief to the lumbar pain (MACDONALD).

With regard to the administration of opium in any form, the rule is, *not to give it when there is suppression, or tendency to suppression, of urine*. Restlessness and sleeplessness are best met by chlorodyne, remembering that the effect of yellow fever on the system is to make it sensitive to narcotics.

The congestion of the kidneys, about the fourth or fifth day, requires watching, so as to diminish the chances of suppression, by reducing congestion and preventing the closure of the uriniferous tubes by accumulated epithelium. For this purpose Dr. Lawson recommends cupping, either dry or with the abstraction of blood, and the use of frictions, with stimulating liniments over the loins. These, with warm baths or hot-air baths, deserve a full trial; and small doses of *acetate of ammonia*, with *potash* or *soda*, or their salts in common use, with diaphoretics (so as to act gently on both kidneys and skin), may prove beneficial.

[Prevention of Yellow Fever.]—Although the profession is not at one respecting the indigenous or the exotic origin of yellow fever epidemics in our Southern States, experience during the late war conclusively shows that both the domestic and foreign factors of the disease may be controlled, and the spread of an outbreak from supposed local causes prevented, or all risk from importation avoided. These questions were fairly tried and decided in and about New Orleans, in the seasons of 1862–63–64–65. Dr. Elisha Harris, in a recent paper *On Yellow Fever on the Atlantic Coast and at the South during the War* (*U. S. Sanitary Commission Memoirs*, 1868), observes: "All the physical conditions that are supposed to promote the prevalence of yellow fever—excepting only such as are immediately controllable by a sanitary police—prevailed continu-

and then add gradually the other ingredients. The dose is from 5 to 10 minims, repeated as often as its sedative effects subside (Compare Mr. Squire's *Companion to the Pharmacopœia*, p. 58, and Mr. Ashburner, in *Lancet* for 11th June, 1864, p. 688).

ally and in unusual force in the delta of the Mississippi during this period of immunity from that disease" (p. 253). Previous to the war the annual average of yellow fever victims in New Orleans was about 1000. The average annual death-rate, from all causes, from 1829 to 1861 was six and a half per cent. There were years when the death-rate exceeded ten per cent. During the aforementioned years, the town was full of unacclimated persons. "One hundred thousand Northern men annually arrived in or passed through, without a single individual being smitten with yellow fever" (HARRIS, *l. c.*, p. 256). These summers passed without a sign of yellow fever epidemic. Besides the observance of a rigid quarantine, the strictest sanitary regulations were enforced by the military government, and to them is to be ascribed the exemption. The thorough cleansing of the towns of Savannah and Charleston, after their occupation by the U. S. troops, and the rigorous sanitary regulations maintained, together with a *military* quarantine, have kept them, it is believed, from any outbreak of yellow fever since 1864.

In the Army Report, already quoted from, Dr. Woodward holds this language: "In conclusion, a few words may be said with regard to the relations of the facts set forth in this report, and in the appended documents, to the prevention of yellow fever. It is to be regretted that the experience of the army throws no more satisfactory light on the treatment of the disease, but it must be admitted that it is most instructive with regard to measures of prevention. Besides those general hygienic precautions which are so important in the prevention or mitigation of all epidemic diseases, two simple and effective measures would appear to be specially indicated by the experience of the army during the war and subsequently. The first is quarantine, as a means of preventing the introduction of the disease; the second is the prompt movement of the command to some rural site on the appearance of the fever among the citizens of the town at which it is stationed, or even after the disease has appeared among the men of the command itself.

"With regard to quarantine, it is well known that a great difference of opinion exists among civil physicians; nor is this surprising, since, in populous cities, approached by many routes of travel, a foreign disease may readily be imported by persons eluding an imperfect quarantine. In such a case it may be quite impossible for the physicians of the place to determine the circumstances, naturally concealed by those who have broken the laws or regulations on the subject.

"In the case of military detachments, however, especially during times of peace, the movements of individuals being so much more readily known, the mode in which such diseases are introduced can very generally be recognized; and hence it is not surprising that recent distinguished English writers on subjects connected with military medicine—Dr. Aitken, in his *Practice of Medicine*, and Dr. Parkes, in his *Hygiene*—are advocates of the doctrine of importation. Dr. Parkes expresses the opinion that the incubative period is longer than is usually supposed, probably often fourteen or sixteen days. Several facts set forth in the appended documents would seem to show that, in certain cases, the disease may be delayed as long as three weeks after exposure. The minimum period of an effective quarantine against yellow fever may then be set down at about twenty days. Twenty-five or thirty days would be better if attainable.

"Should the disease, unhappily, be introduced through neglect to provide an efficient quarantine, it becomes the imperative duty of the medical officer to recommend the immediate removal of the command to some

healthy rural site. On this subject the reports here discussed are explicit. At the only places at which any large number of cases occurred during 1867—at Galveston, at Houston, at Hempstead, at New Orleans, at Fort Jefferson—the troops faced the pestilence, and at each the greater portion of those exposed were attacked. On the other hand, the troops moved on the approach of the disease to camp in the country, escaped almost wholly at New Iberia, Baton Rouge, Alexandria, Shreveport, &c.; while at Indianola, Mobile, Pass Christian, &c., the command being moved after the disease had appeared among the men, almost all those thus removed escaped. The Newbern epidemic afforded a similar experience. After the disease had fairly broken out among the troops, the greater part of them were moved away from the town, and nearly all thus moved escaped. When a command is thus moved, it should be encamped on a dry and elevated site, not previously used for camping purposes; the men should be sheltered by tents, should not be crowded, and should be surrounded by the best hygienic conditions attainable.

"The views here expressed agree with the opinions formed by the Surgeon-General upon the basis of the army experience of 1867 and of previous years. Accordingly, on the 15th of April, 1868, he recommended to the commanding general of the army that a quarantine should be established along the entire Southern seaboard as early as the 1st of May of the present year, and advised the prompt removal of troops from points threatened with infection" (p. xxxviii-xxxix).]

SECTION III.—THE LITTORAL, MALARIAL, OR PALUDAL FEVERS.

Characterized by one or other of these names, three varieties or forms of fever are understood to exist, having many essential features in common. These are *intermittent fever* or *ague*, *remittent fever*, and that variety of *yellow fever* characterized by periodicity of febrile recurrence. The specific or contagious form of *yellow fever* is now understood to be a different fever from remittent and intermittent fever, and it has been considered separately as a continued fever *sui generis*. The malarious form of *yellow fever* is, on the other hand, the same in kind as the intermittent and remittent fevers, but varies greatly in the extremes of its severity. So great, indeed, are the differences induced by the common malarious poison, that "if any one had seen only the milder forms of *remittent fever*, and had no opportunity of tracing up its several grades, he might well believe, when he saw suddenly the severest variety, that he had before him a distinct affection" (ALISON). Such a belief is entertained by not a few. All of the three fevers, however, which are now about to be considered are similar pathologically; while all take their origin from terrestrial aeriform emanations, which are sometimes rendered more active or dangerous in connection with human beings congregated together, and in certain relations as to physical climate, and particularly as to temperature.

[It would appear from the published statistics of the Surgeon-General's Office (*Circular No. 6*, 1865), that during the first two years of the civil war, there were in the U. S. army 262,807 cases of intermittent fever and 1788 deaths; a large proportion of the fatal cases were from the conges-

tive or pernicious form of the disease. During the year ending June 30, 1862, more than one-fourth of the army suffered from these affections. For the same period, there were 123,763 cases of remittent fever, and 1537 deaths. Malarial fevers prevailed to a great extent amongst the Confederate troops, and, in certain localities, the efficiency of entire commands was greatly impaired. In an average command of 878 men, stationed about the forts near Savannah, nearly one-half, on an average, were entered on the sick list each month, and the new cases per month was 220. Throughout a period of fifteen months more than one-fourth the command was unfit for duty, and during the autumn months more than one-half the garrison was on an average incapable of performing military duty. During a period of nineteen months, January, 1862, to July, 1863, inclusive, there were reported in the Confederate army serving along the coast of South Carolina, Georgia, and Florida, with a mean strength of 25,723 officers and men, 157,013 cases of disease and wounds, and of this number 41,526 cases were recorded under the head of some form of malarial fever—a little more than one-sixth, or 16.3 per cent. of all the sickness. The per cent. of malarial disease in the commands was 161, or each man on an average had been entered upon the sick list with paludal fever 1.6 times. In the Confederate forces serving along the coast of the Gulf of Mexico, each man on an average was entered on the sick list, with one or another form of malarial fever, a little over twice in eighteen months. In the (Southern) army of Tennessee, the monthly ratio of cases of malarial fever to the mean strength of officers and men ranged from 4.2 to 17.9 per cent. Even the army of Stonewall Jackson, serving in the Valley of Virginia, an elevated and healthy region, suffered greatly from malarial fevers; for, during a period of ten months, with an average mean strength of 15,582 officers and men, out of 53,198 cases of disease and wounds entered on the field reports, 3876 are recorded under the heads of congestive, intermittent, and remittent fever (J. JONES, *U. S. Sanitary Commission Memoirs*, 1868).]

Pathology.—In these forms of fever a malarial poison of an unknown kind, generated in paludal regions or littoral districts, is absorbed, and affects the blood, as cholera, typhus, and other miasmatic poisons do. The poison, in the absence of any better name, is known as "*malaria*;" and as physicians have merely inferred the existence of such a poison, no exact knowledge has yet been obtained as to its nature and source. Indeed, it still remains to be shown that *malaria* have a substantial existence. No poisonous principle has yet been chemically demonstrated in the air of malarious regions. But many other acknowledged disease poisons are in a similar predicament as to proofs of their substantial existence; and the general impression with regard to *malaria* is, that it is presumed to exist as a gaseous fluid in the atmosphere of certain regions.

After a period of latency, more or less long, functional disorders of the great nervous centres are brought about, terminating in the phenomena either of intermitting, remitting, or yellow fever. These fevers may exist without any alteration of structure being set up, and the patient often dies from the severest forms, with hardly a trace of disease being discoverable. In the milder forms of these fevers, however, a greater number of organs and tissues are morbidly altered than perhaps in any other disease, as the liver,

spleen, lungs, heart, brain, and the serous and mucous membranes of the body generally. The specific action of the malarial poison, within certain limits, may be said to be in the inverse ratio of the intensity of the fever which attends its action. The affections of the liver and spleen also vary greatly according to the country; for in some parts of India the spleen is the organ chiefly affected, while in other districts it is the liver; the nature of the country, perhaps of the soil, impressing evidently some peculiar character on the poison.

The patients laboring under intermittent fever in this country generally recover under medical treatment, without any manifest derangement either of structure or of function of any organ or tissue. When, however, the disease is neglected, the liver may suffer, the disordered function of that organ being generally indicated by jaundice; or inflammation of the liver may ensue, of which jaundice may or may not be a symptom; and this inflammation may be acute or chronic, diffuse or limited to one place. If a liver, previously healthy, becomes the seat of diffuse inflammation, it is of the deepest hepatic tint, and loaded with blood; and we find it often greatly hypertrophied, filling the abdominal and pelvic cavities, and, according as the inflammation is acute or chronic, either greatly indurated or so softened as to be easily broken down. In a few instances this inflammation may terminate in abscess, generally of the usual phlegmonous character. On the contrary, if the liver be previously diseased, its color, even when the seat of abscess, or otherwise most acutely inflamed, may be of the palest yellow, and its texture sometimes so soft and broken down that the larger blood-vessels may be dissected out with the fingers, or so indurated as to form a shapeless mass of varying magnitude. When abscess forms, it may rupture into the duodenum, or into the cavity of the abdomen, or it may point externally.

The paludal poisoning also often produces structural alteration of the spleen. In these cases that organ has been found sometimes so enlarged as to weigh from ten to thirty pounds, greatly exceeding the liver in size (ague cake), while in other cases it is sometimes even less than natural.

[In 161 cases, Dr. Piorry found the spleen to exceed its healthy size in 154. It is said to happen more frequently after tertians and quartans, than quotidians. When inconsiderable, any increase in size can be only ascertained by percussion; but when the organ extends below the costal margin, it is readily detected by palpation. It sometimes stretches to the umbilicus, and may reach to the crest of the ilium. The healthy dimensions of the spleen, given by Dr. Piorry, are: vertical diameter, from $3\frac{1}{2}$ to $3\frac{3}{4}$ inches; transverse, 3 inches. The increase of size is usually proportionate in all its dimensions.]

In consistency, also, it varies from a state of almost fluidity, a mere bag of blood, to a hardened mass with a distinct indurated edge. It is sometimes the seat of abscess; or its parenchyma is transformed into a soft mass of pigment of an extremely dark color.

The functions of the peritoneum may be alone deranged, so as to produce dropsy; but every form of peritoneal inflammation may precede or accompany the *ascites*,—as the serous or the purulent, with diffuse or partial local adhesions.

These are the most usual alterations of function and of structure in the mild paludal fevers of the present day; and in estimating the relative frequency of these secondary affections, *ascites* is the most common, then *jaundice*; while *peritonitis*, *hepatitis*, and *splenitis* are less frequent, and occur, perhaps, in nearly equal proportions.

The pathological phenomena which attend severe intermittent and remittent fever are much more severe, and extend over a greater number of organs. The information afforded us by the dissections of Davis, and the observations of Sir Gilbert Blane, in the cases of the Walcheren remittent; of Jackson in those of the West Indies; of Burnett in the Mediterranean, enable us to understand at least the tendency of the morbid action. Sir Gilbert Blane, in his observations on the Walcheren fever, remarks that the structural derangements were more frequent (especially swelling of the liver and spleen), which then occurred in a very few weeks. Such results seldom occur in England, except under a long continuance of the disease, or after frequent relapses. The morbid changes also extended to the mucous membrane of the stomach, which, in a few instances, was inflamed and ulcerated, and the ulcers had generally a sharp perpendicular edge, as if made with a punch. In cases which died dysenteric, the large intestines, and more particularly the sigmoid flexure and the rectum, were always much contracted, thickened, inflamed, and ulcerated; the ulcers being often so numerous and so confluent that the whole inner surface of the gut appeared in a state of granulation. There is a marked tendency in the phenomena of these paludal fevers to become inflammatory, the congestion of some organ proceeding at once to exudation from the blood-vessels into its parenchyma, which appears to be the cause of prostration and of fatal results. "The significant term *bilious*," writes Sir Ranald Martin, "as applied to these fevers of the East is not an accidental or a misapplied term, as modern statistics fully show. A severe disturbance of the hepatic function is almost universal in the progress of the remittent fevers in the East."

There is another remarkable tendency to be noticed in the persistent effects produced by intermittents—namely, that they impress a character of periodicity to subsequent ailments, especially neuralgic affections; and the disposition to the recurrence of these diseases seems to last for life. Susceptibility to the action of the paludal poison does not diminish, but rather increases by continued residence where it prevails. The returns published by the War Office and Army Medical Department show such a result in the West Indies. Thus, while the annual mortality among the troops resident one year in Jamaica was 77 per 1000 mean strength, in those resident two years it was 87 per 1000, while of those still longer resident it was no less than 93 per 1000.

"In making calculations of efficient force," writes Sir James Macgrigor, "this description of men could not be relied on for opo-

rations long continued in the field" (speaking of men who had suffered from an attack of paludal fever), for "we found that in those who were convalescent or lately recovered from ague, the causes next prone to reproduce the disease were exposure to a shower of rain, or wetting the feet, full exposure to the direct rays of the sun, or to cold, with intemperance, irregularity, or great fatigue." There are many instances also of the same person being repeatedly attacked with the West Indian malarious fever. Sir Ranald Martin writes, with regard to himself, that "after a residence of ten years in Europe, I happened to pass three nights at the best hotel in Strasburg, at a time when ague prevailed in the garrison amongst the French soldiers who had served in Algeria; and two days after quitting the town I was seized with ague at the hour of eleven A. M. (the hour at which ague used to commence with me in India), and I was the only person of the party who was so affected."

The opinion regarding the pathology of *yellow fever*, which holds that it is "*an intense form of the bilious remittent of the tropics*," has given rise to much discussion.

The investigation of this point, however, is attended with extreme difficulties, and is to be carried out with reference to two questions especially,—namely, (1.) The type or mode of progress of the symptoms in mild and severe cases, compared with cases of remittent fever in all grades of severity and stages; (2.) The pathological characters of the morbid processes which take place in severe forms of yellow fever, compared with those of remittent.

Three opinions have thus been held regarding the essential nature of yellow fever. These are,—(1.) That there is a *malarious form of yellow fever* which is an intense and virulent form of remittent, and which becomes more or less a continued fever (CLEGHORN, LIND, HUNTER, ALISON, CRAIGIE, MARTIN). (2.) That it is a continued fever of a specific kind, different from all other continued fevers (CULLEN, CHISHOLM, BLANE, WOOD, HIRSCH, ARNOLD, MACLEAN). (3.) That it is a mixed fever, of a type variable between the remittent and continued forms (JACKSON, MOSELEY).

The grounds upon which the first of these opinions is accepted are—that in the symptoms and effects, progress and pathology, of remittent fevers and ordinary cases of yellow fever, we are unable to discover any essential differences, but merely what is due to intensity of morbid action, degree, and rapidity of progress. Comparisons have been drawn in this way between the summer and autumnal *remittents* of the south of Europe, the *remittent* fever of the Mediterranean, the tropical *remittent* of the East and West Indies and Central Africa, the Bulam fever, or the fever of Sierra Leone and Fernando Po, on the one hand, and between some of the cases of the *yellow fever* of Cadiz, Gibraltar, Malaga, Carthage, Leghorn, Vera Cruz, Havana, Jamaica, St. Domingo, the West Indies generally, and the United States on the other. There are also cases in which no distinction can be drawn between the symptoms, the effects, or the rapidity of action, if the case (considered to be a remittent) is compared with some cases of so-called yellow fever. In other words, it is not possible to distinguish some cases, and say

with certainty that they are cases of remittent rather than of yellow fever, or of yellow fever and not remittent.

[**Chronic Malarial Toxæmia** was very common amongst the United States troops in the late war, who were exposed to the influence of paludal poison. The manifestations of the poison upon the system are slow but characteristic. As observed in the army they have been thus described: The man is evidently out of health, and unfit for duty. He is said to be laboring under "general debility." There is a gradual loss of power, and fatigue comes on from slight exertion, with breathlessness and palpitation; the senses are dull and perverted; there are moroseness, despondency, and irritability; headache, and neuralgic pains in the course of the fifth pair of nerves; lameness of the muscles of the back and legs is often complained of after little exertion; occasionally, there is more or less diminution of sensibility or motion of the lower extremities, which become enlarged, and the integument is shining, smooth, and pits upon pressure; the appetite is capricious and lessened, and there is constipation, alternating with diarrhœa; the urine, at first copious, soon diminishes, with an increase of the urates and phosphates, and frequently of the oxalate of lime, and is loaded with epithelium; the bladder is irritable, with frequent micturition; the skin is harsh, dry, of a greenish-yellow hue and bronzed in portions; and the hair has a dead look and feel. Persons in this condition are very liable to acute disorders, particularly pneumonia, and which constantly are fatal.

Morbid Anatomy of Malarial Toxæmia.—The integument is bronzed, especially, in the regions of the face, neck, sub-axilla, arm, forearm, and outer side of thigh. This change consists in a pigmentary deposit resembling that of Addison's disease, and is to be distinguished from icterus, or the icteroid hue, which is probably from altered hæmatine. Leanness does not amount to emaciation; the fat has largely disappeared, but the muscles retain their fulness. The muscular tissue is generally of reddish-brown color, tears more easily than in its healthy state, but its specific gravity is not lowered. The blood is fluid, but fibrinous coagula full of white corpuscles are found in the cavities of the heart and in the great vessels. The chief and most characteristic changes are, however, to be found in the liver, spleen, kidneys, the lymphatic glands, and the intestinal glandular apparatus. The liver is large, of a pale reddish-slate or fawn-color, and its relative and absolute gravity are increased. It is firm to the touch and divides firmly; the faces of the divided parts are smooth and the edges sharp; the acini are small and indistinct, and the interlobular substance is increased in thickness and development. This increase of the interlobular substance is either by development of its own substance, or, as is more probable, by the addition of new material, albuminous in character. This encroaches upon the vessels, or deposits take place in the walls of the vessels, lessening their calibre and diminishing the supply of blood to the lobules; hence the hepatic cells become pale and shrink, and fatty transformation finally occurs. This fatty metamorphosis I observed but in a single instance; the liver was small and very flaccid. The secretion of bile does not cease; the gall-bladder is well distended, but the character of the bile is changed; it is usually dark brown and tarry in consistence. Corresponding changes occur in the kidneys. They are enlarged, their relative and absolute gravity being increased. They divide firmly. The cortical substance is whitish or fawn, and the cones of Malpighi are congested, purplish, and the papillæ red. The tubules are seen to be crowded with epithelial cells, and their walls

are thickened. From the papillæ may be expressed a milky urine, which is loaded with the *débris* of cells. The change appears to consist primarily in the interstitial deposit of molecular albumen (albuminoid degeneration). Changes, not very definite nor uniform, occur in the supra-renal capsules; the most constant alteration seemed to consist in the relative increase of the cortical substance; but frequently they were not at all changed. The spleen is also somewhat enlarged; its trabeculæ more distinct and tougher; the splenic pulp brick-red, firm and fleshy; the Malpighian bodies, much enlarged, are plainly seen in great numbers throughout the pulp. Very characteristic alterations are found in the intestinal canal. They vary in degree with the stage at which they are examined, but they are always capable of being demonstrated. At the earliest period the intestines are pale and transparent; the solitary glands are slightly enlarged, elevated and filled with a granular matter, albuminous and fatty; the follicles of Lieberkühn come into view by reason of a deposit of pigment in their epithelium, and are thickly strewn at the sites of the oval patches of Peyer; the villi are pale, their cells shrunken, and the basement-membrane transparent (waxy). In the large intestines, a pigment deposit, greenish in color, takes place about the orifices of the tubular glands; the flask-shaped solitary glands enlarge, and their contents accumulate. *Pari passu* with these changes, the veins of the submucous coat become more prominent. The changes in the lymphatic system are found in greatest perfection in the mesentery. Its color is yellowish or fawn, and the glands are enlarged and prominent. Exteriorly the individual glands are red or purplish; interiorly they contain a central whitish, fawn-colored, or yellowish spot of variable size, sometimes solid and resisting, but frequently granular and cheesy. The alterations in the nervous centres are not easily followed. The brain is generally firm, pale, and resists decomposition remarkably. (BARTHOLOW, *U. S. Sanitary Com. Med. Memoirs*, 1867.)

The *morphological changes of the blood in malarial fever* have been studied by Frerichs, and, lately, more thoroughly by Dr. J. Forsyth Meigs, of Philadelphia (*Pennsylvania Hospital Reports*, vol. i, 1868), to whose interesting paper the reader is referred. Pigment matter has been largely found in the blood, brain, spinal-cord, spleen, liver, kidneys, &c. "This adventitious matter occurs in the form of granules, mere molecular dots, very numerous, isolated, loosely aggregated, or connected together in groups (flakes and scales) by a pale substance soluble in acetic acid and in caustic alkalies." The capillaries of the nerve-centres usually contain it in this form (the larger particles not readily passing so far), and the granules often appear as though embedded in the vascular walls. Then again we find these granules contained in cells, not otherwise distinguishable from white blood, or splenic corpuscles in their ordinary condition.* Sometimes these cells have an oblong, or even spindle-shaped outline.† They occur most abundantly in the spleen and portal vein, but appear also, in aggravated cases, throughout the organism. In the liver the molecules are seen adherent to, or contained within the hepatic cells; *i. e.* within their "formed material." Still other, and not less frequent, forms are those of the grain and larger mass; which are distributed in an analogous manner, and are found even in the brain in an extravascular position. Some of these, upon pressure, look like "fragments broken off from yet larger masses." Their size varies indefinitely

[* Pre-existent nucleated cells (of various forms) take up hæmatin, which as the contents of the cells becomes molecular pigment. Rokitansky, *op. cit.*, vol. i, p. 210.]

[† Cell. Pathology. Virchow, p. 257. Frerichs, *op. cit.*, vol. i, p. 320.]

from a mere dot up to grains many times as large as red blood-globules. Frerichs states that some of the masses are $\frac{1}{100}$ of a line in breadth and $\frac{1}{20}$ of a line in length; and these he regards as probable casts of the smaller vessels. My own measurements correspond closely with these. I have not recorded any isolated grains of more than $\frac{1}{200}$ of an inch in breadth, but have frequently observed aggregations of granules and "larger masses" which measured $\frac{5}{100}$ to $\frac{7}{100}$ of an inch; $\frac{12}{100}$ to $\frac{15}{100}$ of an inch was an approximate average size of the separate grains. Now when we bear in mind that the common width of red blood-corpuscles is in man about $\frac{1}{300}$ of a line, and of the white corpuscles $\frac{1}{250}$ of a line, we see that many pigment grains would be necessarily arrested where these could pass. And in different structures of the body the capillaries range only from $\frac{5}{100}$ to $\frac{1}{100}$ of a line in diameter.* In shape these pigment-grains are very irregular, rounded, or sharp, with an angular, brittle-looking outline. Indeed the sharpness of their angles, which, however, are in no degree definite or uniform, "has suggested the term *crystalloid* as applicable to them (Lebert), or rather to the pigment-material common in the lungs."† And I may here allude to their close resemblance to the black matter so frequently seen in old coagula; in patches of chronic inflammation and ulcers of the intestines; and in the fluids vomited in cases of gastric cancer with a bleeding surface. Some writers go so far as to claim an identity,‡ but this is certainly premature. We cannot safely assert more than their common origin in some blood metamorphosis. In color the pigment varies almost as much as in size and shape. Usually it is deep black, quite opaque, and with abrupt, non-translucent margins. Then, again, we find a brown rim through which some light passes. Frequently the color is reddish-brown, or even reddish-yellow, as seen by transmitted light, and the granular scales are seldom opaque. In the splenic pulp I satisfied myself of the existence of red blood-globules in different stages, not only of disintegration, but also of advancing metamorphosis toward black pigment, and am therefore prepared to agree with Frerichs, "that these different colors represent the various stages in the transformation of the red pigment of the blood into melanotic matter." I have occasionally seen tinged gelatinous particles, as though there was a union between the coloring matter and some protein element. But of their intimate chemical nature we are ignorant. All agree, however, that while the pure black forms resist the action of even the strongest acids and alkalies, the paler products lose their color with greater or less rapidity under the influence of these reagents. (MEIGS, *l. c.*, p. 106.)

That the pigment-granules and masses are not foreign bodies, but have their origin in some abnormal action within the organism, and are the product of certain changes of the blood-elements, is probably certain. Whether strictly pathognomonic or not of malarial toxæmia, there is abundant evidence to show their constant connection with that condition, whether acute or chronic. In 90 cases of malarial fever, examined by Dr. Meigs, in 1865, in one only did he fail to discover, in the blood taken from the body during life, pigment-granules. Dr. Meigs gives the following conclusions:

1. That in examining blood during life with a view to determine the presence or absence of pigment-matter, great care is necessary to exclude

[* Human Anatomy, Leidy, Philadelphia, 1861, pp. 337, 339.]

[† Clark, *op. cit.*, p. 611.]

[‡ It is the *pseudo-melanose* or *éléments hématiques* of Lebert; when crystalline the *hæmatoidin* of Virchow.—CLARK.]

all foreign particles from the epiderm or elsewhere. 2. That pigment may exist abundantly in the visceral capillaries, and in the contents of the portal vein and other large vessels, when blood obtained from the derm and subcutaneous tissue does not exhibit it. 3. That only in cases of excessive pigment-development will granules and pigment-cells be visible in such blood. 4. That in the acute stages of malarial fever, the red blood-corpuscles are darker than natural, appear soft, are sometimes crenated, readily yield their coloring material when mingled with water, and are disposed to mass irregularly rather than to form distinct rouleaux. 5. That as the disease progresses they are rapidly and very greatly diminished in number, become pale, lose their tendency to aggregation, and either remain isolated or arrange themselves in imperfect columns. 6. That in the early stage of the disease, the number of white corpuscles is not perceptibly increased, and that later their increase, though very irregular, is always observable; from six to thirty-five appearing in the same microscopic field which presents in normal blood only two or three. 7. That excessive anæmia, with a large increase in the number of white corpuscles, may exist in malarial cases without marked enlargement of the spleen. 8. That no other morphological alterations are to be detected except, as also in some cases of severe anæmia from other causes, certain colorless, highly refracting granules, free or in membranous-looking fragments, appearing like white corpuscles broken up, and giving to portions of the field a filmy, clouded aspect. Thus we see that our diagnosis, except in the most aggravated cases, cannot safely rest upon an examination of blood from the peripheral vessels (*l. c.*, p. 109).]

Causes and Modes of Propagation.—Facts tending to establish the concurrence of certain terrestrial, gaseous, or meteorological phenomena, as necessary to the generation and development of these fevers, are of a very conflicting nature. The concurrence of some, however, are sufficiently obvious, and are applicable to the littoral and paludal fevers generally.

By numerous observations it has been established that some aeriform material of a poisonous nature is exhaled from marshy or wet grounds in the progress of drying. Agues have always been observed to be the diseases of moist or marshy districts; and to prevail most in low, swampy, and humid countries, where seasons of considerable heat occur. The vicinity of marshes, or of a district that has at some recent time been under water; the banks of great lakes, and the shores of great rivers and seas, where the water flows slowly, and in some cases stagnates; in shallow rivers, over land alluvial, low, and flat; extensive flat tracts of wood, where much moisture is constantly present, where the process of drying is uninterrupted, and yet the surface constantly exhaling humidity: these are some of the terrestrial physical conditions in which the paludal and the littoral fevers are found to abound. It must also be admitted, however, that these diseases do not prevail in *all* marshy districts, and they cannot, in some cases, be traced to a residence in the vicinity of marshes. Dr. Wood gives an interesting example of the occurrence of ague from an *irritant cause*, combined with the force of habit. "For seven successive nights M. Brachet bathed, at midnight, in the river Saone, towards the close of October, when the water was cold. Retiring to bed after each bath, and covering himself warmly, considerable reaction took place, which terminated

in perspiration. At the end of the seventh day he ceased to bathe, but was, nevertheless, nightly, about the same hour, attacked with a regular intermittent paroxysm, consisting of the cold, hot, and sweating stages, which returned for about a week, when it ceased spontaneously on the occurrence of an event which kept him out of his bed at the hour of paroxysm, and induced him to take a ride on horseback, which excited and warmed him." Cases having their origin in such causes, however, are of exceeding rare occurrence, so far as the records of medicine show.

The concurrence of circumstances under which paludal and littoral fevers have been observed to become developed may be shortly stated as follows: (1.) A certain degree of heat. A high temperature is especially favorable to the production of malaria, and the more so when acting on moist alluvial soil. (2.) A certain relation as to season, variable with the geography of the locality in which such fevers prevail. The season of the year most marked in tropical climates is that which immediately succeeds the cessation of the rains, or, as it is called, "the drying up of the rains." (3.) Low swampy grounds and extensive rice-fields are well-known sources of malaria. In such districts clouds of mist are often seen, wafted along the earth's surface for miles; and it is believed that malaria, whatever be their nature, cling to such mists. But although it has been observed that absolute marshes do not always produce agues, nor that agues are always due to obvious marshes, yet it is generally found that in districts where such paludal fevers abound the surface is porous, penetrable, and retentive of moisture, although it does not appear on the surface of the ground; that the district had been at one time submerged; and that it continued slowly but constantly to undergo the process of desiccation: or while at certain seasons it imbibes moisture from local or meteorological sources, at other seasons it undergoes the drying process under intense solar heat. Such are some of the most sickly and febriferous districts in Europe, India, and America. For example, the Maremma of Italy; the district of the Lakes near Varna, in Bulgaria; many districts in Burmah; many newly cleared tracts in North America; and many parts in the south of Spain. In most of these places the conditions of the surface of the ground are very much alike. While no obvious appearance of a marsh exists, the vigor of vegetation is extreme, amphibious animals abound, of the batrachian kind, plants and cephalopodous mollusca of notoriously marshy regions find a habitat, and the rich alluvial soil is so imperfectly cultivated that the process of vegetation is not adequately exhausted, and a surface of humid ground is exposed to the solar heat, and so exhales a material which exercises a persistent deleterious influence on the human frame. It is believed that the number of insects and some reptiles with which a place abounds are more significant of its insalubrity than almost any other circumstance; and that a mixture of animal and vegetable matters undergoing decay give rise to miasms much more noxious than those resulting from vegetable matter alone. Dr. Fergusson, in *The Edinburgh Philosophical Transactions*, vol. ix, p. 278, was the first author who clearly proved that the drying of all

porous soils, from which watery fluid readily evaporated, was the genuine source of exhalations capable of producing the paludal fever; and that the febriferous activity of these exhalations was influenced by the character of the season, the moisture, the temperature, and the aerial movements of the atmosphere.

The evidence regarding the geological nature of soil as a cause of ague is somewhat conflicting. It is a fact that the usual localities in which paludal fevers abound are those in which the soil consists of mineral, vegetable, and animal matters, mixed together in such proportions and of such constituents chemically as tend to absorb moisture and retain it, and subsequently to decompose. Such soils are known as *alluvial*. Paludal fevers abound, however, where soils of a different nature predominate. Level plains of sand, or dry, loose, open gravel, are soils where malarial fevers have prevailed.

"The first time I saw intermittent and remittent fever become epidemic in an army," writes Dr. Fergusson, "was in 1794, when, after a very dry and hot summer, our troops in the month of August took up an encampment at Rosendaal, in South Holland. The soil was a level plain of sand, with perfectly dry surface, where no vegetation existed, or *could* exist, but stunted heath plants. On digging, it was universally found percolated with water to within a few inches of the surface, which, so far from being at all putrid, was perfectly potable in all the wells of the camp."

High grounds near exposed marshes are often more unhealthy than the places immediately adjoining, which are on a level with them. Rocky places, such as Ciudad Rodrigo, Gibraltar, and Malaga, have now and then been ravaged by epidemics of littoral and paludal fevers, and the rocky shores and islands of the Mediterranean—for instance, Minorca, Sardinia, Sicily, Cephalonia, and all the Cyclades—abound as much in these fevers as the most level parts of Holland; and the West India Islands, most of which, although coralline rocks, are the native soil of these diseases. Soil composed of tenacious or stiff clay (argillaceous) is highly retentive of moisture, and is difficult either to dry or to drain. The basin of the Thames, comprehending Middlesex, Essex, Surrey, and Kent, is almost entirely clay land, and is the district of England where agues most of all prevail, especially along the banks of the Medway and the Thames. In the days of Sir Gilbert Blane agues had almost entirely ceased to occur in London, and the cases which he treated he believed to have been imported from malarious districts around; and the same may be said of those of the present day. A hundred years before the time of Sir Gilbert Blane, however, we find that agues prevailed in situations in the town of London where they are now wholly unknown, such as Russell Street, Covent Garden, Fleet Street, Fetter Lane, Newgate Street, Paternoster Row, Cheapside, Smithfield, and Fenchurch Street. At the present time ague rarely occurs in London, except on the south side of the river, especially in Bermondsey and Rotherhithe, and chiefly in persons who have recently been exposed to malaria in Kent or Essex, and who have

come from marshy districts, either quite recently or within a few months. The malarious influence, still in the metropolis, seems, however, sufficiently powerful to imprint a periodic character upon various local affections, and occasionally to give rise to fevers of a remittent type. Recently (in 1856) such affections have been unusually prevalent; but the forms of ague now met with in London are more tractable and milder than those which formerly prevailed (Dr. PEACOCK).

It is observed that the surface of the earth may be dried either by the direct rays of the sun, or by currents of hot dry air wafted over it, or by both combined; but it is principally by the direct rays of the sun that the deleterious material of the soil is liberated; and it seems to be at a certain period of this "drying up" process that the exhalations are more potent than at another time in developing paludal fevers. The exposed grounds, after clearing off the copious vegetation from dense jungles, so as to admit the influence of the sun's rays in "drying up," is known to be a fertile source of malaria.

There appears also to be a certain state of the human frame which renders it more than usually susceptible to this disease. The natives of warm and tropical climates are much less frequently and less violently attacked with paludal or littoral fevers than settlers or visitors from other lands, such as the natives of Europe or the northern parts of America. In the Mediterranean, along the coast of Africa, in the East Indies, in the West India Islands, in the Southern States of the Union, new-comers from the northern latitudes are almost invariably attacked, and suffer much more severely from the fever than those who have been long in the country. It has been also noticed that those who, after residing in a territory where paludal fevers abound, have been out of it for some time, an augmented susceptibility to renewed attacks of the fever becomes manifest on their return (CRAIGIE).

Other causes predispose to those fevers, and none more than laborious or fatiguing duty in military or naval operations, laboring in the sun, excess in eating or drinking, intellectual exertion combined with bodily fatigue, and a crowded state of the population. Indeed, *sunstroke*, or heat apoplexy, is regarded by many as a form of remittent fever (JOHNSTON, MARTIN, HILL).

When a remittent fever, or other paludal or littoral fever, has, under certain concurrent circumstances of weather, season, and physical peculiarities, made its appearance in any locality, it necessarily attacks all those who are by constitution, habit, and age, susceptible and predisposed; and the majority of these, especially if enfeebled by previous dynamic or organic disease, it destroys. The population, therefore, which outlives such an epidemic visitation are no longer equally susceptible, and are greatly less likely to be attacked the ensuing season, unless it is more febriferous than the past, which, though sometimes, is not generally the case. The effect of this, therefore, is, that while the endemial disease continues for a season to attack and destroy its *ordinary* annual proportion of the population, it does not for several years attack the *extraordinary* proportion, because that proportion is not yet ready for, or susceptible of,

its attacks. In the course of a few seasons, however, during which the young have grown up and become adult, the adult have become careless, and perhaps irregular and incautious by long immunity, and their constitutions have become less able to resist deleterious or morbid impressions, and the whole population of the place has become generally augmented by the arrival of persons from various other countries; a considerable number of susceptible persons is gradually accumulated; and at the end of five or six years a place of 25,000 or 30,000 inhabitants becomes augmented perhaps by an additional fifth, or even by a third. The majority, or the totality of these persons, are all more or less predisposed and susceptible; a season of excessive drought ensues, in which solar desiccation and little wind form conspicuous characters; fever appears, and spreads at first slowly and gradually, but afterwards, springing up in many points, rapidly coalesces; and in a short time is so general and fatal that it assumes an epidemic character. The usual mortality in the meantime takes place; all the susceptible and predisposed subjects pass through the disease or are cut off; and the population of the place is once more reduced to its state of epidemic insusceptibility and endemic or ordinary liability. This is the usual course of epidemics of paludal or littoral fevers in all countries within the tropics, and, indeed, within the 45th degree of north and south latitude (CRAIGIE).

A question of much interest in connection with *malaria* has given rise to considerable speculation. It relates to the *varieties* of the malarious poison. Is the poison which gives rise to a *quotidian ague* different from that which produces a *tertian*; and is this, in its turn, different from that which produces a *remittent fever*; and this, again, different from that which gives rise to the malarious form of *yellow fever*? Our knowledge is not yet sufficiently precise and extensive to settle these points. But when we see a large body of men placed under the same circumstances as to food, drink, and clothing, and labor, and exposed to the same causes of disease, in the same way and in the same place, some of whom are seized with *quotidian ague*, others with *tertian*, and others with *remittent fever*, the presumption is that the same cause has produced different diseases, according to its intensity, the constitution of the individual, and the predisposing causes to which he may be subjected. Other facts also favor this presumption—namely, *remittent fevers* are known to pass into *quotidian agues*; and these again into *tertian agues*.

AGUE—SYN., INTERMITTENT FEVER.

LATIN, *Febris intermittens*; FRENCH, *Fèvre intermittente*; GERMAN, *Kaltes Fieber*—SYN., *Intermittens*; ITALIAN, *Febbre intermittente*.

Definition.—*Febrile phenomena occurring in paroxysms, and observing a certain regular succession, characterized by unnatural coolness, unnatural heat, and unnatural cutaneous discharge, which prove a temporary crisis, and usher in a remission. These phenomena are developed in an uninterrupted series or succession, more or less regular, which pass into each other by insensible steps.*

Symptoms.—The disease may be sudden in its attack, and without previous illness; but more commonly it is preceded by general indisposition, headache, weariness, pain in the limbs, thirst, loss of appetite, white tongue and frequent pulse, high-colored urine and dark-colored discharge from the bowels. These *prodromes* are accompanied with well-marked exacerbations and remissions of fever, displaying a periodic tendency. After this feverish state has lasted from four days to a fortnight, the patient is seized with severe rigor, and the ague is manifested. The phenomena of an attack or “fit of the ague” are the following:

The paroxysm, like the disease, may be of sudden invasion, and the patient may be in good health up to the time of attack; or it may be preceded by languor, debility, frequent yawnings, and great unwillingness to make the least exertion. In whichever way the cold stage begins, the patient experiences first a sensation of coldness of the extremities, then of the back, and lastly of the whole body; at the same time the nails turn blue, and the features shrink, becoming pale and sharp. If the case be severe, the whole body shrivels up, turns purple, and the surface of the skin assumes that rough condition popularly named “goose-skinned.” The coldness increasing, the motor nerves of the fifth pair are affected, and the teeth begin to chatter; and this tremor extends to every muscle, till the whole body shakes with rigor. Cough, dyspnoea and oppression of the præcordia now occur, with a painful sensation round the temples and down the back. The patient often suffers from nausea and vomiting, and the latter symptom is speedily followed by the hot stage.

[The chill is sometimes so slight as to amount to only a feeling of chilliness along the spine, or over the extremities, or it may be limited to a single limb. The cold stage may be manifested merely by severe pain in the supra-orbital nerve, or there may be simply a drowsiness with excessive yawning or a lethargic state, preceded or accompanied with nausea and vomiting. Dr. Flint has known a state of intense nervousness take the place of the cold stage.]

When the cold stage has lasted a period varying perhaps from half an hour to two hours and a half, a reaction takes place, accompanied by partial warmth, or flushings. These extend, and at length the whole body acquires a heat greater than natural, or from 105° to 107° . As the heat returns, so also does the color, and the body, especially the face, becomes preternaturally swollen and red. The hot stage being formed, the heart and arteries beat with unusual violence, and headache, with a frequent full pulse, and all the distressing symptoms of continued fever, are present. “The mean duration of this stage is from three to eight hours. At its close a gentle moisture breaks out, first on the forehead, and thence extends till the patient lies in a general sweat, sometimes so profuse as to soak the bed and linen as completely as if they had been dipped in water. After the sweat has continued to flow for some time the fever gradually abates, a state of apyrexia ensues, the paroxysm is terminated, and (a sense of exhaustion excepted) the patient feels

restored to health. Sometimes, however, he continues pale, debilitated, and incapable of all exertion, till, on the recurrence of the paroxysm, the symptoms just described are repeated.

Upon the approach of the attack the pulse is slow and feeble, but as the sense of coldness increases it becomes small, rapid, and irregular. When the hot stage forms, the pulse becomes full and strong, and on the sweat breaking out it again becomes soft, less rapid, and at length natural.

The tongue, in mild forms of the disease, is clean in the cold stage, white in the hot stage, and again cleans after the sweat has flowed. In severe cases the tongue is white during all the stages, and also during the apyrexia, while in the worst cases the tongue is brown in all the stages. Excepting some unusual instances, attended throughout with diarrhœa, the patient seldom passes a stool till towards the close of the paroxysm, when it is generally a loose one. It frequently also happens during the cold stage that tumors subside, or ulcers dry up, but the tumor generally reappears, and the ulcers discharge as soon as the sweating stage is formed.

The paroxysm of intermittent fever, of whatever description, is conventionally considered to terminate in twenty-four hours. The duration, however, varies in different types. These types or varieties have been named—*quotidian*, *tertian*, and *quartan*; and of these there are subvarieties—namely, *double tertian*, and *double quartan*. The mean length of a *quotidian* paroxysm is about sixteen hours, that of a *tertian* ten hours, and that of a *quartan* six hours.

The febrile paroxysm, or fit of intermittent fever, has three stages—a cold stage, a hot stage, and a sweating stage. These three stages are not necessarily of an equal duration, but vary greatly in different cases. The duration of the cold stage is from a few minutes to five or six hours, and in general, if the case be severe, the shorter the cold stage the longer the hot stage. The hot stage may last from half an hour to any period less than twenty-four hours. The sweating stage is generally shorter than either of the former, and sometimes does not exist at all. The rule, however, is, that the *quotidian* has the shortest cold stage and the longest hot stage; the *tertian* a longer cold stage and a shorter hot stage than the *quotidian*; while the *quartan* has the longest cold stage and the shortest hot stage of all the varieties. An “*irregular*” variety is also recognized, which takes the form of “*Brow ague*.”

The varieties of intermittent fever are distinguished from each other by the interval of time which elapses between each paroxysm. For instance, when the paroxysm returns every twenty-four hours it is termed a *quotidian*, when every forty-eight hours a *tertian*, and when every seventy-two hours a *quartan*; and these primary types have been extended by early writers to every period comprised within a mensual or bimensual period.

[Varieties in the types are occasionally met with. The *anticipating quotidian* is where the paroxysm, instead of recurring at the usual time, sets in two hours before. The counterpart is the *retarding quotidian*, the paroxysms being put off for two hours (FORDYCE). In the *double*

quotidian there are two daily paroxysms. The *double tertian* is where there are two paroxysms and two intermissions in the forty-eight hours—the alternate paroxysms being similar, while those immediately following one another are not so. In the *triple tertian* there are two paroxysms on the odd, and one on the succeeding days. The true paroxysm in the *double quartan* takes place on one day, a slighter one on the second, while the third is a day of intermission, and there is another paroxysm on the fourth day resembling that of the first, and so on, in succession. In the *triple quartan* there is a daily paroxysm, but it varies on the first, second, and third days,—the paroxysms happening on the first and fourth, on the second and fifth, on the third and sixth, and so on successively, being respectively similar. In the *duplicated quartan* two paroxysms occur on the first day, while there is an intermission during the second and third.

A severe and dangerous variety of intermittent fever is the *congestive*, *pernicious*, or *malignant* intermittent, more frequently met with in the Southern and Western States, and occasionally in those parts of the Northern States where paludal fevers are endemic. During the first two years of the late war, there were reported in our armies 6081 cases of congestive fever, and 1381 deaths, whilst in 156,726 cases of all other varieties of intermittent fever there were only 407 deaths. There are two forms, the *comatose* and *algid*. In the comatose the head-symptoms may vary from lethargy to deep coma; the pulse is large, soft, and generally slow; the respiration is laborious, noisy, and infrequent; the patient lies on his back, unconscious, and cannot be roused; the limbs seem paralyzed; the jaws are locked; deglutition is difficult; there are, sometimes, epileptiform spasms, or active delirium. After the continuance of these symptoms for a variable time, sweating comes on, and there is gradual awakening, with an astonished look, and the senses are regained one by one. The *algid* variety is marked by an icy coldness of the surface, like marble or the collapsed stage of cholera. The extremities, face, and trunk, become cold in succession, the skin of the abdomen remaining warm longest; the tongue is pale, shrunken, and cold; the breath is chilled, and the lips livid. The action of the heart is feeble, and the pulse rapid, small, and almost extinct. The respiration is quickened, broken, and embarrassed. The mind is often undisturbed, and a sensation of repose is felt. The eyes are hollow, glassy, and surrounded with a bluish circle; the face is pinched, and all expression gone. If death does not happen in the fit, the pulse is slowly developed, and the heat of the surface gradually comes back, beginning at the abdomen and extending to the extremities.

The *congestive* variety of intermittent fever is very insidious, and may come on in the second or third paroxysm of a quotidian, or tertian; or it may be initial, though generally preceded by prodromic phenomena, as drowsiness, headache, languor, and gastric derangement. The accession of the *algid* form is sometimes sudden, the patient becoming rapidly cold, lying down, and dying in a few hours. If there should be more than one fit, each succeeding one is more severe. The patient may die in from two to twelve hours.]

Of these primary types it is believed that in Europe the *tertian* is by far the most common type, then the *quartan*, and lastly the *quotidian* (WATSON, COPLAND, CHRISTISON). But this law is by no means general; for M. Maillot treated 2354 cases of intermittent fever occurring in the French army in occupation of a portion of the northern shores of Africa, and he found of that number 1582

were *quotidian*, 730 *tertian*, and 26 *quartan*. In the Peninsular war the *quotidian* was likewise the prevailing type, and at one time they were in the proportion of 16 to 1 of any other type. In the West Indies the *tertian* and the *quartan* are only about one-twelfth of the whole number of intermittents treated, the rest being *quotidians*. At Prome, in Burmah, 298 cases from the 2d Bengal European Regiment were admitted into hospital for intermittent fever in 1853, of which 249, or 83.5 per cent., were *quotidian*, and 49, or 1.6 per cent., were *tertian* (MURCHISON). The results given concerning the Madras Medical Service are very similar (WARING).

[It would appear from the official reports, that of the several forms of intermittent fever, which prevailed in the armies of the United States during the first two years of the late war, that the *quotidian* was somewhat the most frequent form, and next the *tertian*; the *quartan* was rare, and the congestive rarer. Of 262,807 cases reported, 137,038 were *quotidian*, 106,803 *tertian*, 12,885 *quartan*, and 6081 congestive.*

During the months of August, September, October, November, and December, 1863, in the Confederate army serving on the coast of Georgia, and chiefly in and around Savannah, with an average mean strength of 5898, there were 2930 cases of *quotidian* intermittent, 2596 of *tertian*, and 80 of *quartan*, and 9 of congestive (J. JONES, *l. c.*.)]

Most authors who have written on intermittent fever have stated that the accession of the *quotidian* paroxysm occurs early in the morning, that of the *tertian* about noon, and that of the *quartan* in the afternoon, between three and five o'clock. But to this law there are many exceptions. According to Maillot, of 1582 *quotidians*, 1089 occurred from midnight to midday, and 493 from midday to midnight. This result is corroborated by Dr. Murchison's observations at Prome, in Burmah. In 86 out of 113 cases—*i. e.*, in 76 per cent.—the paroxysm commenced between midnight and noon; and in 27 cases, or 24 per cent., between noon and midnight. The most frequent hours of attack were 9, 10, and 11 A.M.; and in 65 per cent. of the cases the paroxysms commenced between 8 A.M. and noon.

Of 730 *tertians*, 550 occurred from midnight to midday, and 180 from midday to midnight; out of 26 *quartans*, 13 were seized from midday to midnight, and 13 from midnight to midday. As the most general conclusion, the paroxysm returned in a great majority of the *quotidian* cases from ten to twelve o'clock, and in the *tertian* from nine to twelve o'clock.

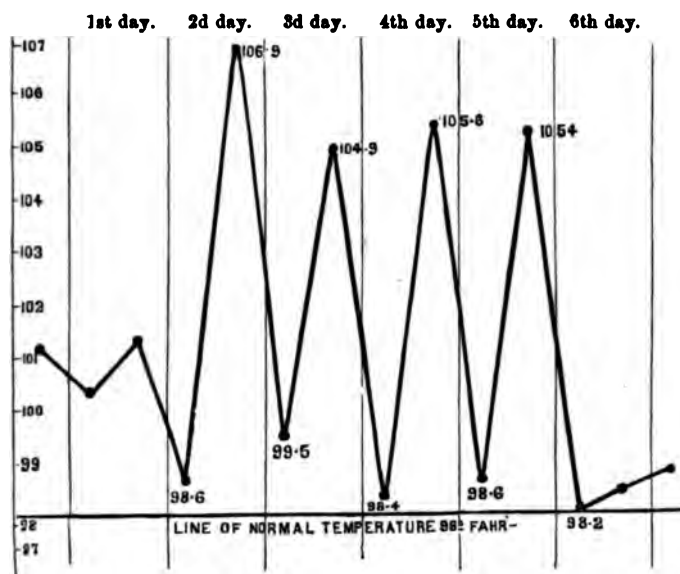
The Temperature in Cases of Intermittent Fever.—The paroxysm of fever, notwithstanding the subjective sensation of chilliness, is invariably indicated by a decided, sudden, and rapid rise of temperature. In this respect it resembles the accession of *febricula*; but while the latter requires only from eighteen to twenty-four hours from the commencement of the rise of temperature to the end of the defervescence, in perfectly normal cases of *intermittent fever* there is

* [Circular No. 6, Surgeon-General's Office, War Department, 1865.]

a whole day free of fever between every two days of the paroxysm. All the types of the fever present this characteristic peculiarity of a sudden and speedy rise of temperature to a high degree (mostly up to 105° or 106.3° Fahr.); and of an equally rapid and complete defervescence, till the period of another fever paroxysm comes about. This comportment as to temperature secures correctness of diagnosis in cases which may be obscure or ambiguous.

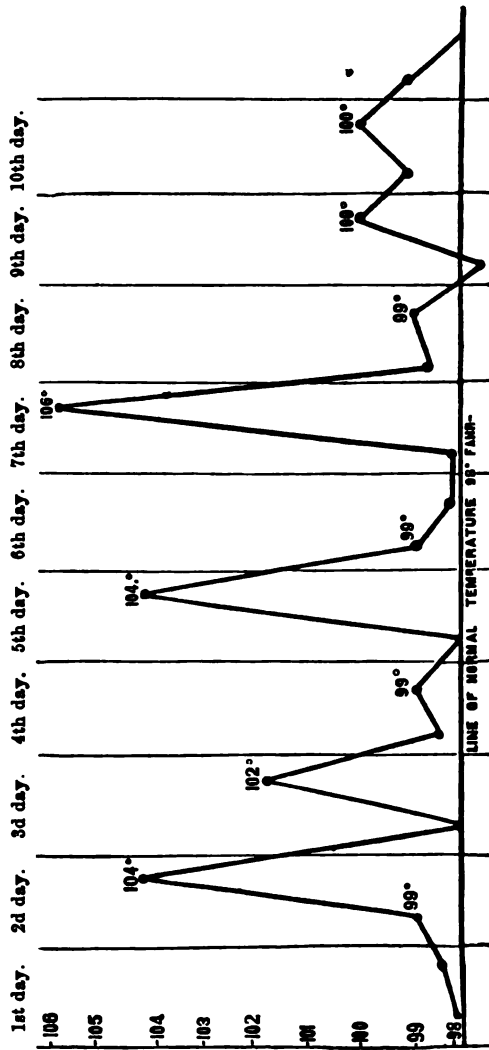
The annexed diagram and that on page 474 represent variations of temperature in cases of malarious fever.

(1.) TYPICAL RANGE OF TEMPERATURE IN A CASE OF INTERMITTENT FEVER OF QUOTIDIAN TYPE. THE RECORDS INDICATE THE HIGHEST AND LOWEST TEMPERATURES DAILY (Wunderlich.)



In a paroxysm of intermittent fever much may be learned as to the relations of the excretions to temperature, and especially those of the urine, by observing the changes of temperature in very short spaces of time: for example, every fifteen, or even every five minutes (MICHAEL, JONES, RINGER). The rise of temperature is found to begin with, or even to precede, the sensation of chilliness. It takes place at first slowly; and gradually, by about the middle of the period of chilliness, the rise becomes greatly accelerated, lasts through the period of the sensation of great heat, and may even extend into the sweating stage. At the commencement of the sweating stage, small vacillations occur, and continue for a short time; and when the sweating has fairly set in, the decrease of temperature begins, and progresses steadily, without any temporary rise, and with great regularity, decreasing at the rate of 2° Fahr. (or more) every five or fifteen minutes, till it has arrived, after several hours, at the normal heat.

(2.) RANGE OF TEMPERATURE IN A CASE OF INTERMITTENT FEVER OF TERTIAN TYPE. THE RECORDS INDICATE THE HIGHEST AND THE LOWEST TEMPERATURE EACH DAY (Wunderlich).



Condition of the Urine.—The observations made on the condition of the urine are divisible into two series, as arranged by Dr. Parkes (*l. c.*, p. 235). (1.) The condition of the urine during the fit, as compared with the urine of a non-febrile period; (2.) The condition of the urine of twenty-four hours during a fever day, as compared with the twenty-four hours' urine of a non-fever day.

During the fit and the apyrectic period the water of the urine is increased in amount during the cold and hot stages: it is most abundant at the termination of the cold or commencement of the hot stage. It decreases during the latter part of the hot stage slowly, and rapidly during the sweating stage. The amount of

increase is variable, and stands in no relation to the quantity of fluid drank, and may be great when this is small (RINGER). The amount of urea excreted by a person with ague, not actually suffering from a fit, is less than in health; but directly the fit commences—that is, at the very first moment of elevation of temperature—or even for some time before this, the urea suddenly increases—an increase which lasts during the cold and hot stages, and then sinks, sometimes gradually, sometimes suddenly, through the sweating stage, or into the commencement of the intermission. It then falls below the healthy average. The amount of increase is very variable, and the type of the fever has no influence upon it; but there seems to be a very close connection between the temperature and the amount of urea. The amount corresponding to a degree of Fahrenheit was greater at a high than a low temperature; and in the fit of each day the same amount was excreted for each degree of temperature (*Med.-Chir. Trans.*, 1859). This increase in the urea must be regarded, in some measure at least, as an indication of increased metamorphosis; and the close relation to the febrile heat certainly implies that it owns only this source, and is not caused by elimination following previous retention (PARKES).

The uric acid is greatly increased during the fit; and after the fit there are often deposits of urates, either spontaneously or on the addition of a drop of acid, and it seems probable that the increase in the excretion of uric acid continues for some time after the paroxysm; and the enlargement of the spleen in connection with this great increase of uric acid is probably not fortuitous (RANKE). The influence of quinine in diminishing the amount of uric acid in health is of interest in connection with its effect upon malaria, and with the condition of the spleen in malarious fever (RANKE, BOSSE). The chloride of sodium is increased during the cold and hot stages to a great degree (TRAUBE, RINGER),—to five times the normal amount; and phosphoric acid is diminished to one-eighth (NICHOLSON).

The results are contradictory regarding the urine of a fever day, compared with the urine of a fever-free day. This may be explained to some extent by the relative duration of the fit, compared with the fever-free period; and great differences may arise from the comparative length of the apyretic period on the fever-days; also from the severity or the reverse of the fit, and from the amount of food and drink able to be taken. With respect to abnormal constituents, albumen is found during the fit in a considerable number of cases. Blood in some quantity, and renal cylinders, are seen about as frequently as albumen; and occasionally chronic Bright's disease is a consequence of ague (PARKES, *l. c.*).

Treatment.—The treatment of agues varies in a great degree with the complications of the disease; such as with the splenic and hepatic congestions, and the inflammatory affections of these and other organs, which are apt to be established during the existence of an intermittent fever. During each paroxysm, and subsequent to it, the condition of the two important organs referred to ought to be carefully observed; and it ought to be observed, also, whether

any symptoms exist of congestion or actual exudation into the cranial or abdominal organs. When the type of the fever is malignant, or of a severe and complex kind, or when the complications are locally severe, it is difficult to cure an ague, which otherwise is a very manageable disease. During the cold stage, especially if it is of long duration, the liver, and especially the spleen, become turgid, the symptoms of which generally disappear with the sweating stage of the fever. It is when the endemic influences are severe, or when the attacks are prolonged over months and years, that these organs begin to suffer permanently from organic disease.

It is useless to attempt the cure of intermittent fever if the sufferer is permitted to remain within the sphere of malarial influences, or even in those geographical latitudes which are said to be peculiarly malarial. It is now an established fact that none can become acclimated so as to withstand the influence of malaria. When organic complications exist, they must, if possible, be remedied, because they maintain the morbid sensibility during the intermission, and prevent the cure of the ague.

In the warmer latitudes the following account of the treatment of intermittent fever is that laid down by Sir Ranald Martin. During the cold stage of the fever, while emetics seem to be indicated, they are not in repute. Warm drinks, ammonia, ether, [chloroform (MERRILL, E. McCLELLAN),] camphor, and other diffusible stimuli, with the application of external warmth, seem to be preferred by most practitioners. During the hot stage a full dose of calomel, with James's powder, should be given at once, and in three hours this should be followed by a brisk cathartic, diluent drinks being freely used meanwhile, along with some cooling diuretic. The tartarized antimony, with nitrate of potash, is recommended, as it answers the double purpose of exciting to action the functions of the skin and the kidneys. On the following morning, the intermission being completely established, the sulphate of quinine is to be administered. The influence of this medicine on a person in health, as observed by Dr. Ranke, is to diminish the quantity of uric acid in the urine (*Med. Times*, May 30, 1857)—an observation which has been recently confirmed by Bosse, of Dorpat, and is of interest in relation to its influence upon the spleen. It is to be given at intervals of three hours during the day, the patient being kept in bed, and supplied with farinaceous food only. In the simple cases, when removed from the sphere of malarial influence, it may not be necessary to give mercurials more than once or twice, but active purgatives are always beneficial in relieving the full and congested state of the abdomen generally, during the continuance of intermittent fever. There are cases of intermittent fever, however, complicated with hepatic and other engorgements, and which continue to recur despite of all means, until a few doses of calomel, followed by purgatives, are administered; then the quinine, which before failed, will speedily cure the disease. The compound jalap powder, combined with calomel, is found very beneficial for this purpose. It appears that certain morbid conditions both of liver and spleen may produce and maintain the tendency to recurrences

of ague. Ramazini relates the case of a patient harassed by an obstinate ague, and who was cured by mercurial frictions administered for syphilis. The influence of splenic disease in keeping up the morbid train of actions of the original fever, and in producing relapses, has been recorded by M. Piorry. In more than 500 cases of ague in which he observed the state of the spleen, he comes to the following conclusions, namely, that the organ is invariably enlarged during the progress of the fever, and that by the use of quinine the spleen diminishes in size; that its reduction in size bears some relation to the quantity of quinine taken; that the effect it produces upon the fever is in proportion to the reduction of the spleen; that the disease is cured simultaneously with the subsidence of the splenic enlargement; and that the fever is apt to recur so long as the spleen exceeds its normal size.

When the fever is severe, accompanied with præcordial oppression, pain, fulness of the spleen or liver, or both, or where there is severe headache, or headache with giddiness, or an oppressive fulness of the chest, a general or a local bloodletting, or both combined, is imperatively demanded, as a means of promoting cure and preventing future evils. The antiperiodic power of bark, quinine, or arsenic, then becomes more easily developed. According to Dr. Copland, such depletion is almost an indispensable preliminary to the administration of quinine or bark, especially in the complicated and congestive forms of the disease. Without such depletion the medicine will either not be retained, or, if retained, it will convert congestions or slight forms of inflammatory irritation into active inflammation or serious structural changes. It is chiefly to a neglect of such a mode of practice that unfavorable consequences have so often followed the use of bark, quinine, or arsenic; for their influence is at first to interrupt secretion, or to over-excite, and subsequently to inflame organs, already loaded, obstructed, and congested. But if blood is to be drawn at all, it should be drawn at the very onset of the hot stage, or that of reaction; and it should be regulated by the constitution, the age, and the habit of the patient, as already explained.

When, on the contrary, the fever assumes a low adynamic form, or when the patient is anæmic, mercurials must be carefully avoided in the treatment under all circumstances, and reliance placed on change of air, quinine, and chalybeates, and improved diet. With regard to liver complication in such cases, the nitro-muriatic acid is to be used internally, in doses of ten drops, three, four, or five times a day, and externally in the form of baths.

With regard to the doses of quinine, some give very large quantities, such as twenty or thirty grains before the expected paroxysm (MAILLOT); others begin to administer the quinine on the subsiding of the paroxysm, and during the sweating stage. According to the experience of Sir Ranald Martin (which has been great in tropical climates), the most rational plan is to give the quinine every three or four hours during the interval of freedom from fever, and in such doses as the urgency of the symptoms may demand. It is to be administered in solution, dissolved by a small quantity of *dilute* sulphuric acid. He also recommends antimony to be conjoined with

the quinine in plethoric subjects; and, on the contrary, if the patient is feeble, irritable, or exhausted, he adds a few drops of tincture of opium to the antiperiodic. When arsenic is given in large doses, and its use prolonged, it permanently injures the circulating system and the mucous membranes of the stomach and bowels. It should be given in small doses, and not persevered in for more than eight or ten days. From six to eight drops of the solution of the College formula may be given every three hours during the interval of freedom from fever. Dr. Murchison's experience in Burmah goes to prove that the practice most effectual in at once checking the paroxysms of intermittent fever is that of administering *one large dose of quinine during the third or sweating stage*. The usual dose given was twenty grains in a draught, with a few drops of sulphuric acid to dissolve the quinine. In no case did he observe any unpleasant symptoms from the physiological action of the drug, although many of the patients complained of slight buzzing in the ears—some amount of which is deemed necessary by Dr. Murchison for the success of the remedy; and when it occurs, "it is a sign that there is no use of pushing the medicine farther." Christison also recommends the administration of large doses of quinine for the cure of tropical intermittent fevers, as deduced from the experience of the medical officers of the Madras army (*Madras Med. Reports*, 1831; *Edin. Med. and Surg. Journal*, Jan. and April, 1855); and Superintending Surgeon Corbyn has long been convinced of the efficacy of this mode of giving quinine (*Indian Annals of Medical Science*, Oct., 1853). Repeated small doses, on the contrary, have been recommended in this country, by many eminent physicians, to be given during the intermissions (HOME, BROWN, BARKER, WATSON); but the evidence of Dr. Murchison is so clear and decided, that one large dose, given as he recommends, seems more efficacious and more economical than repeated small doses.

When the intermittent fever has become chronic, or when there is organic disease of the liver or spleen as a secondary affection, change of climate becomes a measure of necessity, and should never be neglected. Sir Ranald Martin's personal experience does not allow of his writing in favorable terms of the practice of bleeding *in the cold stage of ague*. After quoting many eminent authorities both for and against the practice, he remarks, "that in Europe, at least, the treatment of intermittent fevers by bloodletting in the cold stage, whilst it has the show of being prompt and energetic, proves, in effect, haphazard, systemless, operose, and tedious; and from all that I have seen and heard in the East, the result there has not been more favorable." The rule of practice laid down by Pringle and Cleghorn has received little or no addition in more recent times. Where general bloodletting is had recourse to in the treatment of intermittent fevers, whether simple or complicated, it should, as in the case of all other fevers, be performed at the very outset of the stage of reaction.

[In simple intermittent fever, particularly when there is evidence of derangement of the digestive organs, an emetic of ipecacuanha, or ipecacuanha and sulphate of zinc, should be administered at the outset, followed

by a warm infusion of chamomile, and a mild non-mercurial purgative. Fifteen to twenty grains of the sulphate of quinia should be given in solution, either in a single dose, or in two or three doses at intervals of two or three hours, according to the type and urgency of the case, so that the whole amount shall have been taken two hours before the expected paroxysm. When, from gastric irritability or other causes, quinia cannot be taken by the mouth, it may be administered in an enema, or hypodermically; in the latter case dissolved with tartaric, instead of sulphuric acid, as less locally irritating. Five grains of quinia should be ordered daily, in divided doses, for from fifteen to twenty days, particularly if the disease has lasted any time. It may or may not be combined with iron or arsenic. Where there is intolerance of quinia, arsenious acid, in doses of from one-twentieth to one-thirtieth of a grain, four or five times a day, will be found quite certain. In very young children, arsenic is especially reliable. Numerous substitutes for quinia have been proposed, and have had more or less repute. Amongst these are piperin, tela aranæ (ROBERT JACKSON, CONDIE), bebeerin (LOGAN, WATT, NICHOLSON), ferrocyanuret of iron (STOKES, FLINT), chloride of sodium (J. C. HUTCHINSON), muriate of ammonia, nitric acid (E. S. BAILEY, W. A. HAMMOND), cornus florida, etc. Of these the cornus florida is the most valuable. Recently the sulphites in full doses, have been used, and with alleged success.

The *congestive* variety of intermittent fever requires prompt and vigorous treatment. To save life minutes must be counted. The chief reliance should be on the immediate administration of large doses of quinia—twenty grains, repeated every half hour, or hour, till there is reaction. If the patient cannot swallow, or there is vomiting, it is to be given in an enema, in larger dose. Chloroform alone, or with sulphuric or chloric ether and camphor and capsicum, is a useful adjuvant. Sinapisms should be applied over the chest, abdomen, and to the extremities, or along the spine; or flannels, steeped in hot water to which mustard has been added, and well wrung; or friction with the hands or a woollen cloth. In the *comatose* form dry cups may be applied to the nucha, and along the spine. But the sheet-anchor is quinia; it is the only antidote to the virulent and quickly-killing poison. After the paroxysm is over, if there are no symptoms of cinchonism—and there usually are not—quinia should be given at intervals, in five- or ten-grain doses, and the patient carefully watched, and kept in bed, until the period of recurrence has fully passed. If about the time of the expected paroxysm the nails should become blue and the finger tips shrivelled, or gaping or drowsiness come on, active means must be at once taken.

Chronic malarial toxæmia is best treated by a combination of the chloride of iron, quinine, and arsenic; diuretics; mild saline cathartics, as Seidlitz powders, or the artificial mineral waters—Bitter Kissingen or Pullna; a nourishing mixed diet, including milk, meat, vegetables, and fruit, within the digestive capacity of the stomach; exercise, short of fatigue; and change of climate. Great attention should be paid to the skin; frequent tepid baths should be taken, or the surface sponged daily, followed by gentle and long friction with the hand over the whole body. In some cases the moderate use of *packing*, or the Turkish bath, will be useful. If there is derangement of the chylopoietic viscera, muriate of ammonia alone, or combined with chlorate of potash and colchicum, should be given. Under no circumstances should any preparation of mercury be permitted. Where there is much depression of the nervous system, phosphoric acid, or the hypophosphites, are of great service. In the treatment of malarial toxæmia it is of the first importance to keep the sluices of the system—the kidneys and intestinal canal—open, unless there are contra-

indications; and this is best done by the frequent use of the mild saline cathartics before named. There is great alteration of the blood by the morbid poison: there is an excess of some of its constituents, and a diminished quantity of others; it is imperfect, degenerate, and devitalized; its formative power is weakened, and, as a consequence, the ability of the building and excreting viscera, as well as the functional force of the nerve-centres, are weakened. It must, in a measure, be remade; and elimination through the renal and intestinal organs aid this, by increasing the power and quickening the action of the renewing remedies.*

Chronic enlargement of the spleen frequently subsides as the toxic symptoms abate, and with the general improvement of health. The special treatment is by bromide of potassium, and the application of iodine paint. Professor Maclean, of the Royal Victoria Hospital, Netley, speaks highly of the officinal ointment of the biniodide of mercury. A piece the size of a nutmeg is rubbed in over the affected organ with a spatula. The patient is then directed to sit before the fire until there is a good deal of smarting. About half the quantity is again applied over the tender surface. Some blistering follows, which is to be dressed simply. The process is repeated in about a fortnight or three weeks, according to circumstances. Two or three applications suffice. In no case, he states, has it been inefficacious, and in some its effect has been very striking, reducing rapidly the enlarged organ, and that, too, in very unpromising cases (*Statistical, Sanitary, and Medical Reports (British) Army Medical Department, vol. v, 1865*).]

REMITTENT FEVER.

LATIN, *Febris remittens*—Idem valet, *Febres pestifera singularum regionum*; **FRENCH,** *Fèvre rémittente*; **GERMAN,** *Bösartiges endmisches Fieber*; **ITALIAN,** *Febbre remittente*.

Definition.—*Febrile phenomena with exacerbations and remissions, the remissions being less distinct in proportion to the intensity of the fever. The fever is malarious, and is characterized by great intensity of headache, the pain darting with a sense of tension across the forehead. It is accompanied by functional disturbance of the liver, and frequently by yellowness of the skin. The malignant local fevers of warm climates are usually of this class.*

Symptoms.—There are so many grades of intensity in remittent fever (varying as it does from a severe intermittent to malarious yellow fever), and so many different modifications are impressed on it, from the great variety of country by which the poison is generated, that it is extremely difficult to generalize the phenomena.

The severer forms of remittent fever may be preceded by certain premonitory symptoms, such as languor, listlessness, restlessness, or

* [The following will be found good reconstructive remedies:

R. Leptandrin; Ext. Aloes, aa gr. xxx; Ext. Colchici Rad., aa gr. xv; Ext. Nucis Vom. Alcohol, gr. viij; Resinæ Podophylli, gr. iv; Ext. Ipecac fl., ℥ v; Ext. Hyoscyam., gr. xxx. M. Divide in pil. xxx. Sig. One to be taken every night, or every other night.

R. Pulv. Ferri Sulphat., ʒj; Acidi Nitrici (C. P.), fʒj; Quinini Sulph., ʒij; Potass. Citrat., ʒij; Aquæ Cinnam., ad fʒviij.

Rub the iron and acid well together in a mortar; add first the quinine gradually, until dissolved, and then the potash, first dissolved in water. Mix well, filter, and keep in a dark-colored bottle.

Dose. One or two teaspoonfuls three times daily (GADBERRY).—EDITOR.]

chilliness; or there may be a want of appetite, anxiety, lassitude, pain at the epigastrium, pains in the loins and limbs, headache; slow, small, and irregular pulse; coldness of the skin, and chilliness for one or several days before the commencement of the attack: these are symptoms which usher in a short cold stage. But in other cases the attack is sudden, and the patient, for instance, immediately after a hearty dinner, may be seized most unexpectedly with faintness, vertigo, nausea, confusion of thought; and these almost without a rigor, or a very short one, not exceeding half an hour: a hot stage follows, usually of much greater intensity than that which accompanies the worst forms of intermittent fever.

This hot stage, or period of exacerbation, generally commences in the forenoon of the day, or early in the afternoon, subsiding towards evening, or in the early part of the night, the remissions being generally most complete early in the morning. Sometimes, however, the exacerbations come on towards evening, and last all night, the remissions being then most complete in the forenoon; while, in a few cases, there may be two exacerbations in the twenty-four hours; and these cases are generally the most severe. The exacerbation is usually marked by much cerebral affection, as severe headache, a painfully acute state of every sense, an injected state of the conjunctiva, and great action of the carotid arteries. The pulse, varying from 90 to 120, is generally at first full, but is sometimes from the first small, and generally soft and easily compressible. The tongue is dry, with a white and sometimes yellowish fur, and a bad taste in the mouth. There is generally unquenchable thirst, parched lips, tenderness at the epigastrium, and sometimes pain, with increased dulness on percussion, in the region of the liver. These symptoms are frequently accompanied by delirium, sometimes of a violent character. When giddiness is distressing, and proceeds to delirium at an early period, and runs high, a severe form of fever may be expected. In other cases the patient is oppressed with great drowsiness, lethargy, or coma. The stomach also is often the seat of great pain and uneasiness, followed by vomiting, and the matters vomited are either colorless, bilious, or bloody. The duration of this paroxysm varies considerably, and when the disease is mild it may terminate in six or seven hours; but if severe it may last fifteen, twenty-four, thirty-six, or even forty-eight hours; and Dr. John Hunter once saw a case in which there was no remission for seventy-two hours. Inability to sleep is almost constant. The urine is scanty, high-colored, and of high specific gravity (1024 to 1030), acid, not coagulable by heat (MURCHISON). Albumen was tested for by Dr. Murchison in numerous instances, but never detected; and according to Jones's experience in America, it is very rarely present,—a point of difference, if verified, of great importance as a distinction between severe remittents and specific yellow fever (PARKES). In severe remittent fever Jones found the urea increased, and the uric acid lessened till convalescence, when it again increased, and the pigment was also lessened (PARKES, *l. c.*, p. 242). The fever, however, at length remits, sometimes with sweating, but at other times without any sensible increase of perspiration.

The first exacerbation is generally the longest, lasting, in some cases, for twenty or twenty-eight hours; but generally after twelve or sixteen hours the symptoms remit.

The duration of the remission which follows is as various as that of the hot stage. Sometimes it does not last longer than two or three hours; more commonly it extends to six, eight, ten, fifteen, thirty, or even thirty-six hours. The fever then returns, and in some cases assumes a quotidian type, and has an exacerbation every day, and perhaps nearly at the same time, yet more frequently there is no regularity in the times either of its accession or remission.

The second paroxysm is always more severe than the first, if the progress of the fever has not been checked during the remission, and usually neither any cold stage, rigor, nor even chilliness precedes it. On the other hand, all the febrile symptoms run much higher, the skin is hotter, the pulse more frequent, the headache greater, the senses more confused, and the delirium or coma, when that exists, more violent in degree and more sudden in its accession. Delirium, with more or less loss of consciousness, may not supervene till the third or fourth paroxysm; and is of a low wandering character in the asthenic form of the fever. The tongue becomes dry, hard, and brown, or almost black; the teeth covered with brownish scales; and the pulse becomes small and weak. These symptoms sometimes persevere with or without the black vomit, till they terminate perhaps in coma more or less profound, great prostration, *subsultus tendinum*, fetid breath, resembling the odor of a dead body, convulsions, and at length in death. The severe forms of the fever are sometimes accompanied with a yellowish hue of the skin and white of the eyes. The yellowness is said to be less where there is a copious bilious diarrhœa, and where the urine is of a dark yellow-brown color. When the disease does not terminate fatally, amendment is generally observed after the fifth exacerbation, which may subside in very copious perspiration, with the following symptoms of progressive amendment: the tongue begins to clean and grow moist at the edges, the sordes disappear from the teeth, the thirst diminishes, and the appetite gradually returns. The pulse remains slow and soft, but begins to acquire strength; and the skin continues cool and moist, sleep returns, and the strength is gradually but very slowly recovered. Headache may continue for some days, till relieved by epistaxis. Young and robust men, particularly recruits, who recently arrive in India from Europe, suffer a considerable amount of vascular excitement, with marked symptoms of determination of blood, either to the head or to the abdominal viscera, at a very early stage of the fever. In such cases the pulse is at first full, and of tolerable strength, the skin burning, and the delirium raging and acute. Again, in other men more advanced in years, or those debilitated by long service in India, by previous disease, or by habits of intemperance, there is very little vascular excitement, even during the exacerbations; and the pulse, though quick, is small and weak. There is no great heat in such cases—there is even coldness of the skin, with a yellow tinge, often severe hiccough and vomiting, with great prostration of all the vital powers. In such cases the

chief indications of the exacerbations are increased restlessness, vomiting, headache, or wandering delirium. In these cases the remissions are not well marked, even from the commencement of the attack (MURCHISON, *l. c.*).

There are great varieties in the degree of severity and type of this fever, more especially as they occur in England, France, Holland, and Germany, compared with those which occur in Spain, Italy, the Mediterranean Islands—or still more so in Africa and the East and West Indies; and accordingly some authors (CRAIGIE) distinguish three varieties,—*e. g.*, (1.) The autumnal remittents of temperate countries, as England, France, Germany, Holland, Hungary. (2.) The summer and autumn remittents of warm countries, as Spain, Italy, Greece, the Mediterranean coasts and islands generally, the Levant, the north of Africa and Asia, and the United States. (3.) The endemic remittents of hot and tropical climates, as in the south of Asia, Central and Western Africa, Equinoctial America, and the West India Islands. Accordingly, remittent fever has received different names from the localities where it prevails. Thus we have the *gall-sickness* of the Netherlands, the *Walcheren fever*, fever of the Levant (IRVINE), Mediterranean fever (BURNETT), Hungarian sickness, *puka fever* of the East Indies, *jungle fever*, *hill fever* of the East Indies, *bilious remittent* of the West Indies and Mediterranean, *Bulam fever*, *Sierra Leone fever*, fever of Fernando Po and Bight of Benin, *African fever*, and *Bengal fever*. Prevailing on the borders of inland lakes, as in America, it is sometimes called the *lake fever*. (See page 56 on such nomenclature.)

[Remittent fever is, after intermittent, the most prevalent type of fever in the Middle, Southern, and Western regions of the United States. It is their summer and autumnal epidemic, and unacclimated strangers are very liable to be attacked with it in visiting those sections. From its annual presence and severity in so large a portion of the country, its study is of interest and importance to the American physician. It begins very much like a paroxysm of intermittent fever, with a varying period of lassitude, yawning, and pain in the head, back, and limbs, particularly the calves of the legs; an indescribable uneasiness about the stomach sometimes precedes all the other symptoms; the tongue is coated, and there is a bitter taste in the mouth; the pulse is small, and the action of the heart labored, with increased impulse, and intensity of the sounds. The initial chill may be severe, or moderate, or, what is more frequent, there is a general sensation of cold, lasting from fifteen minutes to a couple of hours, during which there is great thirst, with nausea and vomiting before its termination, particularly if a meal has been recently eaten. The hot stage comes on, with increase of the throbbing headache, which is usually frontal, but occasionally occipital. Violent pain in the back is often complained of. In some cases there is wandering delirium, most frequently associated with a drowsy stupor, shown when the patient is half awake, and passing off when he is quite roused. The pulse is quick, rising in the first paroxysm to 120 or 125, and may be small or full, but rarely hard; the tongue is coated with yellowish fur, and dry, though it may remain moist and almost natural in color; the respiration is hurried and sighing; the thirst is excessive; the urine scanty and muddy. The paroxysm lasts from five to ten hours, when a remission takes place. The first may be decided,

and last several hours. In the subsequent exacerbations there is an aggravation of all the symptoms of the first paroxysm, except the chill, which is rarely well marked, though slight shivering, or a sensation of coldness, often precedes the second or third exacerbations. When there is a recurrence of the chill, it is most commonly at the tertian period (STEWARTSON). Dr. Boling says: "Where the fever is of the double tertian type, the first and third, perhaps the fifth, exacerbations may be ushered in by tolerably distinct agues, while the second and fourth may be preceded but by the very slightest sensation of coldness, if any. The pulse in each succeeding paroxysm becomes more frequent, and there is a gradual diminution in fulness. In the subsequent remissions a corresponding increase in frequency will be noticed; and though relatively to the preceding exacerbation the pulse shall have fallen, it will still be quicker than during the preceding remission. In the second exacerbation the moisture about the tongue is slight, though the tongue is not very dry. In the third and fourth it is apt to become dry, at least on the dorsum, the edges yet moist. Still later, it is parched, rough, and cracked. In each succeeding exacerbation it becomes drier, of darker color, contracted, and sharp-pointed, with intensely red edges and tip. During the remissions the dryness and other morbid characters somewhat abate; and in the early remissions it often becomes moist, and otherwise nearly natural. In each remission the tongue assumes a more natural look; but in each succeeding one this is less than in the immediately preceding one—so that in any given remission, though its appearance will have improved from its state during the preceding exacerbation, it will be worse than during any former remission." The salivary secretion, diminished or even suspended during the exacerbations, becomes free during the remissions, but less so with each succeeding one. There is great disgust for food, and craving for cold and sour drinks. Distressing and constant irritability of stomach is constantly present, increasing with each paroxysm. The matter vomited is bitter, and of a yellowish or greenish color, or, later, dark grass green, and small in quantity. In many cases the vomit is, besides what may have been lately swallowed, a tough, glairy fluid, which holds suspended small, dark-green flocculi, which fall to the bottom of the vessel; or there is a greenish-brown, dirty-looking sediment. The bowels are costive, and the evacuations, provoked by purgatives, serous, containing but little fecal matter, and yellow or greenish. Delirium, when it happens in the later exacerbations, is rarely violent, and commonly abates or ceases during the remissions. The mind seems occupied with the ordinary avocations and thoughts. Perspiration during the remissions is less and less marked as the disease advances; and the skin gets a yellowish tint, first noticed in the conjunctivæ, or there may be jaundice. Sudamina are met with in protracted cases; and herpetic vesicles, singly or in groups, seated usually on the edges of the lips, or about the *alaë nasi*, are common (J. F. MEIGS). There is no true eruption. The sense of debility is extreme, and is often as much complained of in the first or second exacerbation as later in the disease, when the actual debility is greater. At whatever time of the day the first exacerbation may happen, the tendency in the subsequent ones is to come on the after part of the day—some time between noon and six o'clock in the evening—and to continue during the night, the pulse rising to 112 or 116; while in the morning it will fall to 100 or 96. In remittent fever of the double tertian type, however, it will, in most cases, be found to occur alternately in the fore and after part of the day. In protracted cases, say from the eighth to the twelfth day, the remissions become less and less marked, not

more so than in continued fever; the distinctive type—periodicity in the exacerbations and remissions—is lost; and ataxic and adynamic symptoms supervene. Bronchitis is a common complication.

A favorable change is earlier indicated by the secretions of the mouth and tongue than by any other sign. Even when the tongue is quite dry in the exacerbation, some moisture is apt to appear upon the edges and lower surface during the remissions; and diminished intensity in the coming exacerbations may be inferred from the slightest increase of this moisture during a remission, upon what it was in a previous one.

The *diagnosis* between remittent and typhoid fever is, in general, very easy, even when the intermissions are not well marked, and it has run into the continued type. The absence of many of the chief distinctive traits of typhoid fever will enable us to avoid an error. In remittent fever there is no true eruption, no constant tenderness, with gurgling on pressure, in the right iliac region; the intelligence is nearly always good in the beginning, and may continue so throughout; and the peculiar besotted expression of the face in typhoid is wanting. The access is much more sudden. There is usually an initial chill.

Malignant congestive or pernicious remittent fever (African fever, Country fever, Lake fever), styled by Dr. Dickson "a hideous and pestilential modification," prevails in our Southern, Western, and Northwestern States. It begins often as a simple intermittent, and the first paroxysm attracts but little notice. The next chill is more severe; there is extreme coldness of the surface, which is shrivelled, and of a livid hue, and the body is bathed in a clammy sweat, sometimes limited to the face and neck. There is violent gastro-intestinal irritation, with incessant purging and vomiting, the discharges being often mixed with blood, and rarely with bile; the intestinal evacuations have been described as having the appearance of water in which a piece of recently-killed beef has been washed (PARRY). The abdominal tenderness is slight, but a sense of weight and burning heat in the stomach are complained of. The thirst is intense and unquenchable. The respiration is difficult and peculiar—a deep-drawn double inspiration, or double sigh, and one expiration. The pulse is small, thready, and frequent—120 to 140 beats in a minute; but, according to Dr. Boling, the action of the heart continues strong, as shown by the loudness of its sounds and the force of its impulse. There is excessive restlessness, the patient tossing about, and wanting to get out of bed. The intelligence may remain good during the attack, though there is sometimes delirium, and coma may come on after the second paroxysm. Severe headache is very constantly present. If the termination is happy, the restlessness abates, the skin dries, the temperature of the body slowly rises, and the pulse becomes slower and fuller.

A *comatose* variety is met with in the Southern States, which resembles very much the same form of intermittent fever already described. Stupor comes on in the first paroxysm after the cold fit, with dilated pupils and stertorous breathing. As it declines, the stupor passes off, and there is no alarming symptom during the remission. In the exacerbation, the lethargic state returns more marked, and may at once deepen into coma; and this is repeated until recovery or death. The remissions are sometimes very imperfect, and marked only by a temporary and slight abatement in the force, and a diminution of a few beats in the frequency, of the pulse, and cessation of stertor, with yawning and stretching. Dr. Boling relates a case where the patient lay eight days comatose, waking up during the hour of remission on the ninth morning.

The *anatomical character* of remittent fever is a peculiar alteration in

the color of the liver, which is more or less uniformly bronze, or a mixture of bronze and olive, or some shades of lead color, the natural reddish-brown being lost, or only faintly to be seen (STEWARDSON, SWETT, HOWARD, POWERS, ANDERSON, FRICK, STILLÉ). This essential hue is caused by the deposit within the liver-cells of hematoïdin, and described by Frerichs as pigment-liver (A. CLARK, J. F. MEIGS). Prof. Alonzo Clark found, in two persons who had had remittent fever several years before their death from other diseases, the same color of the liver, less intense than in recent cases, but yet well-marked, and the microscope showed the coloring matter unchanged, except, perhaps, in quantity. (See page 461 of this volume.) The liver is often softened, and the gall-bladder distended with thick grumous bile, resembling molasses. The glands of Brunner are enlarged (STEWARDSON, FRICK, ANDERSON, STILLÉ). The glands of Peyer are often slightly prominent, and their mucous membrane injected and softened. The spleen is enlarged and softened, and the heart flabby.

Treatment.—With fever so various in its degrees of severity, it is not possible to do more than indicate the nature of the treatment which may be followed, as every special case must be prescribed for and treated by its own special indications, and with a due regard to the nature of the prevailing epidemic.

The extent to which bloodletting can be carried, as recommended by Drs. Irving and Cartan and Mr. Goodison, will depend on the constitution of the patient, the type of the fever, the season, the climate, its immediate effect, and whether the prevailing epidemic is of such a kind as to be benefited by bloodletting. From the testimony of Dr. Hennen as to Corfu, Mr. Muir as to Cephalonia, Mr. Goodison as to Zante, and Mr. Boyle as to Sierra Leone, those who have long resided in these places do not bear bloodletting so well as strangers from colder and more temperate regions. When bloodletting is beneficial, its effect is in general to abate remarkably the pain, throbbing, and constriction of the head, and the pain of the orbits, to relieve epigastric oppression and tenderness, to render the pulse slower, less tense and oppressed, and to render the motion of the blood more free and less embarrassed. In some instances in which delirium is urgent, leeches or cups applied to the occipital region are of the greatest benefit.

Local depletion over the epigastric region is often of great service, and enables the stomach to retain fluids and medicine. Purgatives are indicated to unload the alimentary canal, and to relieve the congestion of the visceral bloodvessels. The form most useful is the compound powder of jalap, with calomel, given in a bolus, and followed by three or four ounces of infusion of senna. Sometimes ordinary doses of purgatives have little effect till the local depletion has been effected over the region of the stomach; and it is also a good plan to change the purgative from time to time.

In every form and variety of the fever one of the most important guides in the treatment is to be derived from the nature of the prevailing disease, whether endemic or epidemic. Too much attention cannot be given to every means of knowing the type of the epidemic fever, whether *sthenic* or *asthenic*, and to study each individual case in relation to the prevailing type. First, the *duration of the stage of*

the fever must be ascertained,—*i.e.*, whether it be of some hours' or of some days' duration, and whether, when the practitioner sees the patient for the first time, the actually existing paroxysm is at its accession or its decline. It is known by experience that the means of treatment which would be salutary during the first few days cannot be used later to the same effect and in the same amount. There is less tolerance of remedies, and their effects are less therapeutic. Again, it is also known that the means which would arrest fever and save life, if applied at the accession of the paroxysm, would induce a dangerous collapse, or even destroy life, if applied at the stage of its decline, or towards its termination.

The various therapeutic agents which have been employed with various degrees of success in the treatment of remittent fevers are—*emetics, the warm bath, tepid and cold affusions, cold drinks, bloodletting, purgatives, diaphoretics, mercury, quinine or bark, arsenic, wine, and opium*. A review of the prominent modes of treatment of remittent fever, by the most eminent of British army surgeons, has led Sir Ranald Martin to make the following general remark: namely, that a disease so varying in its nature, so general and complicated in its influence on the system, is not to be justly treated by one remedy. Bark and calomel, each a remedy of great power, will nevertheless not succeed in the cure of fever if used exclusively; and so it is with the most powerful of all means, bloodletting. Each remedy must therefore have its proper place in the treatment.

The first and most immediate object of treatment is to reduce the force and frequency of arterial action during the paroxysm. If the patient be seen in the forenoon of the first, second, or third paroxysm of an ordinary remittent fever of *sthenic type*, and if he is of a sound constitution, and not beyond middle life, bloodletting from the arm, while the patient is in the recumbent posture, should be practised to the extent of relieving the sufferer from præcordial oppression, from visceral fulness and congestion, or from the intensity of the headache, whichever may predominate. The quantity of blood to be taken is to be regulated by the effects produced, and not by any arbitrary measure in ounces. Evidence of relief from visceral congestion is obtained from the following indications: namely, reduced force and frequency of the pulse, reduction of morbid temperature, and gentle relaxation of the skin. This relaxation of the skin ought not to proceed to sweating, with further symptoms of depression of the vital powers. If it should do so from untoward circumstances, from half a grain to a grain of opium, or from fifteen to twenty drops of laudanum, with as many of chloric ether, should be administered, the object of the administration of either of these medicines being to influence and soothe the heart's action, and to allay gastric or intestinal irritation; and it is only in cases of depression that opium is to be administered thus early in the treatment of the fever.

One general bloodletting will generally be found sufficient to relieve the patient from abdominal or cerebral oppression; and it will further have the effect of simplifying and rendering more efficient.

all the subsequent means of cure.* Within an hour after the bleeding, a dose of *calomel*, with *compound extract of colocynth* and *James's powder*, should be given, followed in two hours by a powerful cathartic, such as *infusion of senna* with *sulphate of magnesia*; [or from ten to twenty grains of a pilular mass composed of resin of jalap and powdered rhubarb, eight parts each, and *calomel* and quinine, four parts each.] After the free action of these remedies, some degree of remission will be obtained in the afternoon, and the patient should be directed to take at bedtime from three to five or six grains of *calomel*, with four of *James's powder*, if the skin be dry; and during the past eight or ten hours he may have the free use of cooling drinks. On the early morning visit of the following day the remission will probably be more complete, when the *sulphate of quinia* alone, or in combination with the purging mixture, should be freely and repeatedly administered. Sir Ranald Martin recommends that it be given *with* the purgative mixture. By the forenoon the paroxysm may again recur in a milder degree, though to such an extent as to demand the application of leeches to the epigastric region, if any oppression or uneasiness exist there, or behind the ears if headache persist. A mixture composed of *antimonial wine*, with the *acetate* or *nitrate of potash*, should be given every two hours, so as to soften the skin and determine increased action of the kidneys. By these measures the daily decline of the disease is seen, and consequently there is a daily diminishing occasion for the use of active measures of cure, till, towards the fifth, sixth, or ninth day, convalescence is established.

If, however, remittent fever has existed unrestrained for several days, and the patient has not been seen till the accession of the third or fourth paroxysm, or even later, a general bloodletting is still the principal means of saving life, *provided the general powers of the constitution remain uninjured*; and it is to be followed by *calomel* purgatives, and quinine, in the manner already indicated.

If the paroxysms have become indistinct, running into each other, with brief or ill-defined intervals, while abdominal or cerebral complications arise, as indicated by epigastric fulness, or by approaching stupor or delirium, *bloodletting* may even now constitute the principal means to save life; but the blood *must be still more gradually abstracted* than before, whether generally or locally. Generally speaking, it is to be done by leeches, at the accession of the paroxysm. *Antimonials* are also to be used; cold must be applied to the shaved head; and while sinapisms and blisters must also be applied, on the influence of *calomel* chief reliance is to be placed, and the very first dawn of remission is to be seized upon to give quinine. We are not, in such cases, to wait for a clean tongue, the absence of heat of skin, or local complication. It must be given every *three* or *four* hours, with an occasional mild aperient in the intervals, until the dangerous symptoms shall have yielded—a result often observed to be coincident with the manifestations of the mercurial influence.

* [In this country general bloodletting is unnecessary, and often harmful.—EDITOR.]

Dangerous symptoms, such as those just noticed, will sometimes rise suddenly, without any loss of time on the part of the medical attendant, or neglect in treatment. If such symptoms are associated with yellowness of the skin, in persons broken in health, or of feeble constitution, or of dissipated habits of life, or who may have undergone much mental distress, the chances of a fatal termination are imminent.

When the spleen is enlarged, mercury is not to be used in the treatment of the fever; and bloodletting, either general or local, is not borne well. The blood is changed in such cases; it is more or less dissolved, and a general cachexia prevails.

The period of convalescence demands no less careful attention on the part of the medical attendant, especially as to diet and a timely removal from all malarious influences, by a voyage to sea or a change of climate. It is to the mismanagement of convalescence, and a too early discharge from hospital principally, that we must refer the numerous and fatal relapses in the fevers and dysenteries of our seamen and soldiers (MARTIN).

Regarding the method of treatment just described, my friend and colleague, Professor Maclean, writes in the following terms:

"I have been led to take a view of the treatment of malarial fevers generally, and remittent fever in particular, differing from that laid down by many authors. It appears to me that the so-called antiphlogistic treatment, so much insisted on by many writers, is based on the belief that the phenomena observed in a case of remittent fever are consequent on a process of inflammation. It is only on such a belief that antiphlogistic treatment can be justified.

"During the exacerbation of a remittent fever there is violent disturbance both of the vascular and nervous system. Almost every organ, almost every function suffers,—the gastric intestinal membrane is affected, the liver and spleen suffer, the brain is involved, for rending headache and delirium are often present. Is it rational to suppose that an inflammatory process can be going on at one and the same time in all these various organs? Do the appearances observed post-mortem give any support to such a doctrine? If not, on what principle can spoliative treatment be justified? Is it not rather the case that this terrible disturbance of so many organs is due to the presence in the blood of a subtle poison acting on them all? If so, surely the guiding principle of the physician in his treatment should be to counteract this poison, to neutralize it, or to expel it from the system, and so to prevent a recurrence of the exacerbation. This is the principle on which I have long acted, and I am satisfied that it is at once a safe and successful one. In quinine we have such an antidote—a therapeutic agent of unrivalled efficacy, which, if skilfully used, will rarely disappoint the expectations of the practitioner.

"It is always, of course, advisable to have the bowels thoroughly evacuated; and if the patient is seen when his stomach is loaded, it is well to evacuate its contents by an emetic. In ardent remittents, however, there is generally little call for this, as obstinate vomiting is almost always a troublesome symptom. This done, the period of remission must be watched for, and, the moment it arrives, quinine in a *full dose* should be given—*not less than fifteen grains in the case of an adult*. If

the irritability of stomach be so urgent that the remedy is rejected, while measures must be adopted to allay it—such, for example, as alkaline remedies in combination with hydrocyanic acid, turpentine stupes, or even a blister to the epigastrium—time—precious time—should not be lost. *Quinine* should be given *by the rectum in a full and efficient dose*. By mouth or by rectum, or by both, quinine, in quantity sufficient to induce some of the symptoms of saturation (cinchonism), should be given before the time of expected exacerbation. According to my judgment and experience, it is bad practice to withhold quinine until an impression has been made on the force and frequency of the heart's action, from fear of increasing headache, causing congestion of organs, or the like. An impression on the force and frequency of the heart's action is best attained by arresting the paroxysm; and this is done most quickly, simply, and effectively by the early administration of quinine. I have over and over again had patients brought to me from the malarial quarters of the city of Hyderabad, in whom it was impossible to distinguish any period of remission—the tongue black and dry, sordes on the teeth, the skin hot and parched, the pulse enormously quick, the intelligence feeble or gone—all pointing to a system so charged with malarial poison as to be well-nigh overwhelmed. In such cases quinine, with concentrated beef-tea and brandy, are urgently called for, and should be administered freely; and it is astonishing how men, by such measures, are often snatched from impending death. I have seen in a few hours consciousness return, a striking reduction in temperature, in the *frequency* of the pulse, with a remarkable accession of force and volume, follow the treatment indicated above. I do not advise, and never used quinine in the heroic doses advised by some. I have never exceeded a \mathfrak{D} ; but within such reasonable limits I have never seen it aggravate headache. On the contrary, I believe that in remittent fever—in fact, in all forms of malarial fever with which I am acquainted—I believe quinine to be a powerful remedy in quieting the tumultuous action of the circulation disturbed by the presence of this terrestrial poison. For some years past Warburgh's tincture has been much used in the treatment of malarial fevers in Southern India. It is a secret remedy, and therefore open to the objections very properly urged against all such remedies. It is understood that quinine enters largely into this remedy, and I do not doubt it. Be this as it may, I have given this 'tincture' a fair trial in some of the gravest forms of malarial fever, and it has also been extensively used by some of the most experienced officers of the Madras army; and I do not hesitate to say that I think it a valuable remedy. I have known it arrest at once some of the severest cases of remittent fever, no exacerbation appearing after the second dose. It almost invariably acts as a powerful diaphoretic—the most powerful with which I am acquainted. I have seen patients, under the influence of this remedy, saturate not only the bed-clothes but the very mattress, the patient's room and his person for days after giving out a strong odor of the medicine. For this reason it requires to be used with extreme caution, if at all, in the adynamic form of the disease. In urgent cases I follow the practice of the American physicians. I do not wait for a remission, but give quinine at once; and in all I am conservative of the patient's strength. I have seen violent delirium follow free leeching of the temples, and over and over again seen extreme and dangerous prostration follow depletive treatment, and that in cases where the violence of the disturbance indicated *power*; but these signs of power in the system are often most delusive, and, if combated by depressing measures, we must be prepared for sudden signs of collapse. Against

the system of treating this fever by saturating the system with mercury I enter my strenuous protest. I know nothing more deplorable than the condition of a patient whose constitution, already depressed by the presence of malaria, is further saturated by another poison which acts as a powerful ally of the first."

In the asthenic form of remittent fever, such as that so well described by Dr. Murchison as prevailing in Burmah, it is necessary to exercise great caution in depletion. All the cases he relates which had been freely bled exhibited the most aggravated typhoid symptoms, and most of them died. Even in the instance of young and robust recruits, low adynamic typhoid symptoms were sure to supervene in a short time after bloodletting; and, even although it gave temporary relief, it was certain to aggravate, if not to induce, the subsequent typhoid condition. If the headache is very severe and the pulse full, a few leeches may be applied to the temples at the commencement of the attack; but if the hair be cut short, or shaved off the scalp, cold lotions applied to the head, or the cold douche kept up for ten minutes at a time, gives great relief, and is the preferable remedy (MURCHISON). As soon as possible after the commencement of the paroxysm the bowels should be cleaned out with a purgative of calomel and compound jalap powder; or by colocynth, antimonial powder, and calomel. If typhoid symptoms betray themselves, stimulants, such as wine and brandy, must be given; but, as in intermittent fever, "quinine is undoubtedly the sheet-anchor," and it is best given, as in the former fever, in one large dose of twenty grains at the very commencement of a remission. Carbo-azotic or picric acid has been lately introduced as an active remedy in the treatment of malarious fever. Prepared by Calvert, of Manchester, it is of a light yellow color; and in doses of two grains, cautiously repeated, it is to be pushed till the patient gets yellow-skinned.

MALARIOUS YELLOW FEVER—SYN., FEBRIS ICTERODES REMITTENS.

Definition.—*Febrile phenomena due to malaria, in which the exacerbation and remission are so connected that the fever resembles a continued fever, and is characterized by great intensity of headache and yellowness of the skin* (COPLAND, DICKENSON, BOOTT); *but in which the urine is not suppressed, and continues free from blood or albumen.*

Pathology.—It immediately results from the history of yellow fever, that in its malarious form it is the product of the coasts of the West India Islands, the American equinoctial continents, several districts in Spain, and the west coast of Africa. All over the Caribbean Sea the disease takes place sporadically, or in insulated cases every season, more or less numerous according to the subjects and the number of new visitors, and there never is a season in which a few cases do not occur. At Vera Cruz, Havana, and other towns on the Spanish Main, malarious yellow fever invariably attacks Europeans or Canadians who may land there between the months

of May or June and October or November; but so long as such cases continue few, isolated, and sporadic, they attract no attention, and the disease is not heard of in ordinary years. It seems to prevail, for the most part, in towns situated on the sea or river coasts of alluvial countries in warm climates; and that, while the banks of these rivers or seas are liable to occasional alternate periods of inundation and drying up, the fluctuations of the tides, co-operating with these, contribute powerfully, under intense solar heat and a windless atmosphere, to render the towns along the shores of such districts the seat of malarious yellow fever. "While ague is the offspring of the marsh or its margins, and remittent is the effect of a more concentrated form of the same exhalation from some moist surface in the process of solar desiccation, the malarious form of yellow fever appears to be the product of that state of the atmosphere which takes place after a long continuance of solar heat, with little or no wind, in those points chiefly where the atmosphere of the sea and that of the land are in constant communication and interchange. It is, indeed, a remarkable fact that the intense form of remittent fever which has been distinguished as malarious yellow fever, and sometimes as 'bilious remittent of malignant type,' is rather rare in the interior of countries, and is seldom found in towns, situated on rivers, higher than the influx of the tide. The fevers which appear in these situations are more of the usual remittent character; and in the interior of the American continent there is little doubt that the *lake fever* represents the malarious *yellow fever* of the coasts. Even in Europe, while the towns on the sea-coast and on rivers were laboring under the malarious yellow fever, the sickness in the interior approached more to that of the remittent or remittent continuous type" (CRAIGIE). For this reason the term *littoral*, as well as *paludal*, is used to designate this class of fevers.

The endemic conditions under which the malaria give rise to this form of yellow fever might be referred,—(1.) To thermometric temperature of the air; (2.) To the state of the atmosphere as to currents of winds and electricity; (3.) Local peculiarities of surface already referred to; (4.) Constitutional susceptibility, and crowding together of masses of people.

That intense solar heat contributes greatly to the development of the yellow form of malarious fever, is shown by the situations of those parts where it is peculiarly endemic, in relation to the prevailing temperature. Thus, it is found to prevail chiefly in places situated in the eastern regions between 10° of south latitude and 42° of north latitude. On the continent of Europe it has generally prevailed in places situated between the 36° and 38° north latitude, and has never gone farther north than Barcelona on land, or in latitude 48° north on the sea. That it has gone farther north, it has been alleged, but the authenticity of the statement is doubted. In these northern latitudes it is also observed that the malaria of yellow fever cannot pass over a thousand yards of water without being deprived of its power.

The following observation was made by Sir John Pringle on the fevers of Walcheren and South Beveland in 1747:

"These epidemic fevers, by reason of the great heats of the season, not only began more early than usual, but were fully as fatal to the natives as to us. But Commodore Mitchell's squadron, which lay all this time at anchor in the channel between South Beveland and Walcheren, in both of which places the distempers raged, was neither afflicted with fever nor flux, but amid all that sickness enjoyed perfect health—a proof," he says, "that the moist and putrid air of the marshes was dissipated or corrected before it could reach them" (*Diseases of the Army*, p. 58).

The very same observation was made at the very same spot, fifty-two years afterward, by Sir Gilbert Blane:

"I had, in the course of this service (at Walcheren, in 1809), an opportunity of observing the extent to which the noxious exhalations extended, which was found to be less than I believe is generally known. Not only the crews of the ships in the Road of Flushing were entirely free from this endemic, but also the guard ships stationed in the narrow channel between this island and South Beveland. The width of this channel is about six thousand feet; and although some of the ships lay much nearer to the one shore than the other, there was no instance of any of their officers or crew being taken ill with the same disorder as that with which the troops on shore were affected" (*Med.-Chir. Trans.*, vol. iii, p. 27).

It is now also generally believed that the malarious form of yellow fever cannot exist except in places where the average range of temperature is high throughout a considerable part of the year; and for this reason it is believed that it will not become a disease of this country. Sir Gilbert Blane asserted that it never appeared either in tropical climates or in the temperate latitudes, unless when the atmospheric heat has been for some time steadily at or above 80° Fahr., 21° of Reaumur, or 26.67° Cent.; according to Humboldt, 75° of Fahr., or 24° Cent.; and according to Matthei, 72° Fahr., or more. The disease is also found not to prevail in mountainous situations. According to Humboldt, it has never ascended to 3044 feet above the level of the sea, and according to Sir Ranald Martin, never above 2500 feet; and below the former limit the Mexican oaks do not flourish, showing that the constant average temperature below this is of a tropical character. In Jamaica, according to Dr. Craigie, it rarely ascends 1600 feet above the level of the sea (Dr. Lawson's instance of the outbreak at Newcastle being considered an instance of "specific yellow fever"). "In Jamaica the medium temperature of Spanish Town in the hottest months is about 85° Fahr., or between 83° and 85°; and in Kingston it is much the same, ranging from 85° to 90°, and rarely falling below 80° from May to the end of September. At the more elevated parts, however, the temperature diminishes, being only about 70° at Stony Hill, elevated about 1300 feet; at Cold Spring, 4200 feet above the level of the sea, only 60°; and at the summit of the Blue Mountains, which are estimated to be 7200 feet above the level of the sea, the thermometer is found

to range in August from 47° at sunrise to 58° (HUNTER) at noon: or at an average of 60° (MOSELEY).

At Stony Hill, the first of these places, yellow fever has sometimes, though not very often, displayed its epidemic virulence in a very bad form.

In the Island of Trinidad, however, the ridge behind Port of Spain, which is a limestone rock elevated 1500 feet above the level of the sea, has been highly productive of yellow fever, and has cost the lives of many men in attempting its clearing and fortification.

The composition of the soil has been believed to exercise some considerable influence on the production of malarious yellow fever. On this subject, however, the facts are discordant. Alluvial soils are those where malarious yellow fevers have mostly prevailed, as at Grenada, St. Domingo, New Orleans, Philadelphia, New York, Boston; or calcareous, as in Jamaica. It has also been observed that a beach, bank, quay, or wharf is the place where the disease first makes its appearance, when such beach, bank, quay, or wharf is alternately immersed in sea-water and exposed to the drying effects of great solar heat. The drying effects of great solar heat have also been supposed to extricate some deleterious material from the green wood of new ships (WILSON), and also from forests of mangroves (INGRAM, HUMBOLDT, WILSON).

In all those localities where the disease is endemic it seems to manifest a decided preference for the natives of the colder regions. Thus the British, Germans, Swedes, Danes, are more liable to suffer than Italians, French, or Spaniards; and in ordinary years the natives, and especially the colored population, are rarely attacked.

The diagnosis between the specific contagious yellow fever and the malarious form of yellow fever is at all times difficult; and severe marsh fevers in certain geographical limits have a close resemblance to contagious yellow fever. But they are not contagious, and urinary and blood symptoms do not occur in them. It must also be remembered, in accounting for its origin in any case, that marsh fevers have become developed weeks, and even months, after exposure to the exciting cause.

With regard to the further history of the phenomena and treatment of this form of fever, the reader is referred again to what is said under remittent fever, and simply stating that when an observer has seen only the milder form of marsh remittent fever, and is then suddenly called upon to witness an attack of malarious yellow fever, he may well believe that the affections are entirely distinct. But after a time, when the intermediate forms have been more closely scrutinized, it is found that at no point can any valid line of demarcation be drawn between the several forms of these malarious fevers, so numerous are the connecting links which bind them to each other (ALISON, PARKES).

[TYPHO-MALARIAL FEVER.—*Chickahominy Fever, American Camp Fever.*
(DR. CLYMER.)

Definition.—*An idiopathic fever of mixed type, caused by a combination of paludal and pythogenetic influences, with marked remissions and exacerbations at the beginning, and, after a variable period, becoming continuous; attended with early prostration, diarrhœa, and subsequently extreme adynamia: the characteristic lesion is enlargement and ulceration of the solitary intestinal glands.*

History.—This form of fever attracted attention first in 1862, as the Chickahominy fever, from its prevalence in the Army of the Potomac at that time, but has since been common whenever our armies operated in malarious regions, amongst men saturated with paludal poison, exhausted by over-exertion and insufficient rest, imperfectly nourished, exposed to the action of animal effluvia from the decaying bodies of both men and brutes, and drinking water impregnated with the products of common putrefaction. These coincident causes, tending to lower the vital forces and corrupt the blood, produce a compound disorder, in which the combined action of paludal, pythogenetic, and scorbutic influences are evident, and which varies in type, as one or other of the determining conditions is predominant. The name *typho-malarial* was proposed and first used by Dr. J. J. Woodward, U. S. A. (*Outlines of the Chief Camp Diseases of the United States Armies*: 1863).

Symptoms.—The attack is generally sudden, beginning with a chill; there are headache, anorexia, thirst, diarrhœa, and sometimes epistaxis. The tongue soon becomes coated with a thick, dry, brown fur. For some days there are distinct remissions and exacerbations; in the early part of the second week they become less marked, though they may persist throughout the attack. Regular remissions very commonly again take place on the approach of convalescence. Diarrhœa is apt to be troublesome and persistent, as the continued type is developed; the mouth is coated with sordes; an herpetic eruption may appear about the lips and nose; wakefulness is constant, with low muttering delirium; tympany, rare in the first week or ten days, occurs; and purpuric blotches, or petechial spots, with hemorrhage from the bowels, gums, mouth, and nostrils; and all the phenomena of a low form of fever now set in. Matter resembling coffee-grounds is sometimes vomited towards the last. If the disease is to terminate favorably, regular remissions again happen, generally in the forenoon, with evening exacerbations. Congestive pneumonia, bronchitis, and parotitis are the intercurrent affections, particularly bronchitis. An attack lasts from three to five weeks; and convalescence is very lengthened.

Anatomical Characters.—The characteristic lesion is enlargement of the solitary follicles of the small intestines. There may be universal congestion of the mucous membrane of the small intestines, more marked in their lower part, or there may be only congestive patches of variable size in the ileum, the solitary follicles being enlarged from the size of a pin's head to that of a pea, and black with pigment deposit; they sometimes look like yellow mustard-seed sprinkled on a red ground; their apexes are sometimes ulcerated. The mucous membrane of the colon may be of a slate color, with patches of congestion and spots of ecchymosis, or it may be streaked of ash and dark red. Small ulcers are occasionally found in the follicles of the colon, cæcum, and appendix vermiformis. The patches of Peyer are generally unaltered, though they may be con-

gested, of a dark red hue and slightly prominent, and the individual follicles forming the agminate patch may be the seat of a pigment deposit giving the "shaven-beard" look, or bluish-black tattooing, the adjacent membrane being uncongested. The spleen is usually enlarged, and most frequently softened, though its texture may be firm. The lower lobes of the lungs and bronchial mucous membrane are congested (WOODWARD).

Treatment.—Quinia should be given by the mouth, in enema, or hypodermically, according to circumstances. It will not, no matter how promptly given and in what dose, cut the disease short, in a very large majority of cases, if it ever does; but in moderate, and fractional, doses—10 to 15 grains in the course of the day—it decidedly modifies and controls the disorder by its antidotal property. When, later in the attack, marked remissions reappear, it may be administered in larger doses with advantage. The treatment generally should be that already directed for typhoid fever.]

SECTION IV.—MUCOUS FEVERS.

Under this heading it is proposed to group together and to consider the remaining diseases of the *miasmatic* order which are to be noticed in this text-book, and which (with the exception of *Croup* and *Dysentery*) are by some comprehended amongst the "*General Diseases*." These are,—*Influenza*, *Whooping-cough*, *Mumps*, *Diphtheria*, *Croup*, *Dysentery*, *Diarrhœa*, *Cholera*.

These diseases are all attended with fever, and are characterized by irritation, catarrh, inflammation, specific lesions, or altered functions of some portion of the mucous membrane, either of the respiratory or alimentary tracts.

INFLUENZA.

LATIN, *Catarrhus epidemicus*; **FRENCH**, *Grippe*; **GERMAN**, *Grippe*—**SYN.**, *Influenza*; **ITALIAN**, *Influenza*.

Definition.—A specific febrile disease, invariable in its essential characteristics, frequently prevailing as an epidemic, attended with lassitude and prostration to an extreme degree, with special and early implication of the naso-laryngo-bronchial mucous membrane; chills and great sensibility to cold over the surface of the skin, the eyes injected and tending to fill with tears, the nostrils discharging an acrid fluid, attended with fixed and intense pain in the head, mostly frontal over the eyes, sometimes also attended with giddiness; nights sleepless, with delirium or lethargy; cough prevails, with yellow expectoration, most troublesome at night, and tending greatly to increase the headache. Fever attends the disorder, sometimes slight and sometimes severe, and of a type varying in different epidemics and localities. The duration of the fever is definite, of from four to eight days (PARKES). The sense of taste is generally greatly disordered, and there is great anxiety and depression over the region of the heart.

Historical Notice.—We have no credible accounts of the existence of influenza previous to the tenth century. In 1311 it was very fatal throughout France. In 1403 the courts of law in Paris were closed on account of the deaths. Towards the close of the twelfth and thirteenth centuries it was observed that catarrh was not only

endemic in particular districts, but that it occasionally spread over large portions of country, while still later, in the year 1557, it was found to prevail epidemically, not only over the whole of Europe, but even over the whole of the northern hemisphere, beginning in Asia and proceeding westward till it terminated in America. In the eighteenth century, having advanced westward till it reached the Elbe, it passed over the intermediate countries and reached England, where the stream broke into two branches, the one crossing the Atlantic to America, while the other retrograded southeast through France, Spain, and Italy, till it was lost in the Mediterranean—a course similar to that described by cholera.

Influenza has occasionally originated as far eastward as India, but more commonly it has broken out in the north of Europe, as Moscow, Warsaw, or Dresden. It seems probable that, like the poison of Cholera Indica, its spread may be limited to a small number of primary *foci*; for we find in every volume of the *Calcutta Transactions* accounts of some catarrhal fever spreading for a season along the banks of some principal river, and then subsiding; so that it is evidently only occasionally and at long intervals *erratic*, as in 1729, 1743, 1775, 1782, 1831, 1833, and 1837. The influenza, therefore, is both endemic and epidemic; and, in the latter case, we find it, at least in Europe, spreading from east to west, prevailing in the depths of winter as well as the heights of summer, lasting nearly the same space of time in the different towns and cities it attacks, or from four to six weeks, affecting contiguous places in different degrees and at different times.

On looking to the habits of this poison, it is probable that its actions are not limited to man; for in most years, when influenza has been epidemic, a similar disease has been epizootic, especially among horses and dogs, as in the years 1728, 1732, and 1775. It is a disease of extraordinary rapidity of progress; and as its diffusibility is great, so are its periods of recurrence frequent—those cycles of its visitation which are as yet beyond our comprehension to explain.

Pathology.—A specific poison is believed to be absorbed, and to infect the blood, when, after a period of incubation varying from one to two or three days, or even to two or three weeks, it produces disordered functions of the great nervous centres, causing great general depression, extreme debility, together with slight or severe remittent fever. The specific actions of this poison are on the mucous membrane of the eyes, of the nose, and of the bronchi; in a smaller number of cases on the mucous membrane of the fauces, causing sore throat; and in a still smaller ratio on the substance of the lungs and on the pleura, causing inflammation of those organs. In most instances the disorder terminates in diarrhœa. These different pathological phenomena vary in frequency and complexity in different seasons and places.

In most cases, where the poison is of sufficient intensity to produce fever, the type is remittent in this country, with exacerbations in the evening. Its usual duration is two, three, or four days, when it terminates in an abundant sweat, and which not unfrequently

leaves great debility behind it. In Germany the fever is sometimes intermittent. At the same time, however, or, it may be, preceding or succeeding the fever, the patient has in general been seized with a slight inflammation of the ocular and nasal membranes, followed by coryza, or the serous discharge of a common cold or catarrh; and this inflammation generally extends to the larynx and trachea, or to the lungs.

The pneumonia occupied most commonly the middle and lower lobes, and only rarely the summits of the lungs. Out of forty cases observed by M. Landau, the inflammation occupied twenty-one times both lungs, eleven times the right lung, and eight times the left. The forms of pneumonia are principally serous inflammation and red hepatization, the latter occasionally interspersed with a few points of pus. Gluge states that in the fatal cases of pneumonia connected with influenza he has found exudations in the bronchia, which he can only compare to the false membrane of croup. Such exudations were seen in the hepatized portions of the lung as white, elastic, firm cylinders filling the bronchia, from the fourth or fifth divisions of these tubes, into such as are not more than a quarter of a line in diameter. The inner membrane of the bronchia in such cases was extremely reddened, but not softened.

Symptoms, Course, and Complications.—The symptoms of influenza assume a variety of different forms. Thus, catarrh often exists without the fever, and in a smaller number of cases, the fever without the catarrh. Severe nervous depression, prostration, anxiety, and præcordial oppression, were frequently the most prominent symptoms, while in other instances the bronchial affection alone harassed the patient.

The disease usually begins suddenly with chilliness and shivering, rapidly succeeded by an immediate and evident impression upon the mucous membrane of the nose, mouth, frontal sinuses, trachea, and bronchial tubes, to a greater or less extent. General soreness accompanies these symptoms, with severe, darting, neuralgic headaches, aching of the limbs, listlessness, great mental depression, complete anorexia, and an extraordinary weakness, which, in the experience of Dr. Parkes, bore a close ratio to the extent of the pulmonary affection, and consequently to the severity of the disease. These symptoms were accompanied by fever, slightly increased towards evening. Patients were usually seen about the third or fourth day, and then they were found complaining of cough, tightness of the chest, of pain in the epigastrium, and also of dyspnoea. The face was flushed, and sometimes swollen, the alæ of the nose red, the lip vesiculated, the eyes streaming with coryza, and the voice altered as in a common cold. The tongue was moist, or coated with a yellow mucus, and taste was vitiated, the skin soft and without morbid heat, the pulse little augmented in frequency. But although each of the particular symptoms might be mild, there was a languor, debility, and dejection of spirits far beyond what might have been expected, and almost exceeding that of common continued fever. These symptoms were in many instances long in subsiding. The average duration of the cases in the epidemic of 1847 (so admir-

ably described by Dr. Peacock) was from three to five days in the mild forms, and from seven to ten in the more severe.

In mild cases such phenomena constituted the whole disease, and the patients recovered about the eighth or tenth day, after suffering for a few hours from sharp diarrhœa or profuse perspiration. In many instances, however, the patient, in addition, suffered from mild or severe sore throat; or cough came on, and continued for many weeks. In a few cases the symptoms were of a more aggravated character, the fever being more marked, the pulse accelerated, the skin hotter, and the cough more troublesome; and these conditions have often been followed by inflammation of the lungs.

The pulmonary complications may be arranged into four forms,—(1.) Capillary bronchitis; (2.) Bronchitis supervening on tuberculous disease of the lungs; (3.) Bronchitis with disease of the heart or aorta; (4.) Pneumonia.

The accession of capillary bronchitis is indicated by the chest symptoms becoming more severe and the cough paroxysmal, and the dyspnœa at first quite disproportionate to the cough and to the physical signs. The expectoration is scanty, and consists of small yellowish pellets, forming tenacious masses of a peculiarly nodulated form. The pulse becomes rapid (120—140), the tongue covered with a white-brown fur, and prostration is extreme. The only auscultatory signs are roughness of the inspiratory murmur, with occasional sibilus, and slight crepitation at the back. There is soreness and contraction of the chest, but no acute pain. Crepitation, unattended by dulness on percussion, soon extends over a greater or less extent of both lungs; and the dyspnœa speedily becomes so intense as to prevent the patient from lying down, the lividity of the lips and face increases, and the eyes become prominent. The cough is now very frequent, the sputa very viscid, of a greenish-yellow color, without air-bells, and often streaked with blood. The respirations are quickened; but there does not appear to be any uniform connection between the extent of the disease and the disturbed ratio of the pulse and respiration movements. The general rule is, that the respirations are relatively more quickened than the pulse (PEACOCK, PARKES). The physical signs soon become modified by rapidly developed emphysema of the lungs. Generally, it may be said that the *capillary bronchitis of influenza* is distinguished from *pneumonia* by the greater severity of the general symptoms; by the tendency of the fine crepitation of the early stage to pass into subcrepitant and mucous râles, rather than to give place to evidences of condensation, and by the peculiar characters of the cough, which is paroxysmal, and not attended by pain; and, lastly, by the character of the expectoration, which consists of whitish viscid pellets, cohering into irregular masses, and destitute of the glairy adhesive character, russet color, and small air-bubbles of pneumonia expectoration (PEACOCK).

Inflammation of the substance of the lungs seldom occurs till the second or third day, and more commonly not till the fifth or sixth; and, although generally, is not always preceded by shivering, or even bronchitis. The pneumonia in some years has been charac-

terized by well-marked symptoms, as pain in the side, dyspnoea, and by purulent or sanguineous expectoration, so that nobody could mistake it; but in general the pneumonia has been adynamic in character, and presented a striking contrast to the usual symptoms, there being scarcely any local pain, the pulse, ordinarily so large and full, has been slow and small, and though sometimes counted between eighty and ninety, has ranged more commonly from sixty to seventy. The face also, instead of being full and red, has been sharp and pale, the lips blue, and the extremities cold. The patients also, who generally preserve a good deal of power in the ordinary forms of pneumonia, were now so weak that they were obliged to be supported while auscultated. Even this mode of exploring the chest did not afford the usual indications, for crepitation was rare, the respiratory murmur was heard, except in a few points, all over the chest, and there was little or no bronchophony. The auscultatory signs are in general those simply of bronchitis, dry rhonchus in some parts, and harsh vesicular murmur in others. The expectoration likewise had not the characters observed in simple pneumonia; for, instead of being purulent and mixed with blood, it was thin, transparent, and viscid, and, if fever prevailed, it was usually of an adynamic character, marked by a brown tongue, an accelerated pulse, and occasionally by delirium. Throughout the progress of this disease the symptoms of nervous derangement are much more prominent than in ordinary catarrh, and the muscular debility is great, which is the most distinguishing feature of the disease. So great is this prostration, that in some instances the patient has fainted merely by attempting to sit up. This extreme debility often continues after all other symptoms have passed away. The disease generally terminates favorably by perspiration, or by a copious secretion of mucus from the bronchia, or a copious discharge of urine, which deposits a sediment on cooling. Towards the termination of the complaint, rheumatic affections, especially of the face and head, assume an intermittent type.

Causes and Modes of Propagation.—Influenza is for the most part so universal a disease that large portions of the population of every country in which it has prevailed, without respect to age, sex, or condition, have been commonly infected. The air seems to be the main medium of propagation, rather than food or drink; and the special agent of propagation seems to be one capable of indefinite increase, continual reproduction to a greater or less extent in different places (PARKES). In general, women, from being less exposed to the weather, have suffered in a smaller proportion than men, and children less than either. In all epidemics the aged suffer greatly. According to Dr. Blakiston's results, the ages from ten to sixty furnish the most patients. The ages from thirty to forty furnish most male patients, and from twenty to thirty most female patients. In the epidemic of 1847 the mortality was greatest amongst the adults and aged. In childhood the average mortality was raised 83 per cent.; in manhood, 104 per cent.; and in old age, 247 per cent.

It has been remarked in several epidemics that the lower parts of towns have been more generally and more severely affected than

the higher and more healthy districts. The epidemic of 1847 was much more fatal in the insalubrious parts of London than in those less unhealthy; and according to Dr. Peacock's experience, the mortality of influenza was owing more to the condition in which the disease found the patient, than to any inherent power of the poison itself—a result conformable to general experience and the returns of the Registrar-General (PARKES).

The nature of the "*epidemic influence*" which gives rise to *influenza* is quite unknown. Sudden changes of temperature appear to assist the development of the influenza poison; and exposure to cold predisposes the individual to the disease—which seems to be a disease especially of the higher latitudes.

Susceptibility Exhausted.—Few persons suffer more than one attack of influenza in the same epidemic, although many relapse; and one attack of this disease in no degree protects the constitution from a second attack in another epidemic.

Prognosis.—Children and persons under forty die in a very small proportion, unless in a previous state of ill-health. The mortality, however, among the aged has in every country been great from this disease. It has been remarked, also, that the disease, if not fatal in itself, left the patient, of whatever age, often greatly debilitated in body and depressed in spirits, and that those with tender lungs who suffered from it frequently fell into phthisis, or continued to cough for several months afterwards, so that a complete recovery was often long and tedious.

Treatment.—As a general rule, the great majority of cases in epidemics of influenza have scarcely required any medical treatment. In that of 1782 it was observed that "many, indeed, were so slightly indisposed as to require little or no medicine; nothing more was wanted to their cure than to abstain for two or three days from animal food and fermented liquors, and to use some soft, diluted, tepid drink. A lenient purgative at the beginning of the disease was useful in moderating the fever, and nature seemed to point out the repetition of it afterwards when there was pain in the stomach and bowels, and a tendency to diarrhœa. The same was observed in 1762. Nothing, likewise, was observed so successfully to mitigate the cough as a gentle purge to open the bowels, and afterwards to give a gentle opiate at night. In the year 1837 it was also remarked that, as long as the symptoms were limited to cough, hoarseness, headache, or other pains moderate in degree, the patients all recovered by putting them on a low diet, by attending to their bowels, and confining them for a few days to the house; and, if more was attempted it was quickly found that the disease ran a course scarcely influenced by medicine. A smaller number, however, required medical attendance, either from the severity of the bronchitis, the occurrence of pneumonia, of angina or of severe dyspnoea, of the disordered state of the bowels, or more frequently from the debility induced by the disorder.

Bloodletting is always hurtful. It does not relieve the fever, and increases the nervous depression. In general, when pleurisy, bronchitis, or pneumonia may supervene, leeches to the chest, or

cupping, may relieve symptoms. A mild purgative dose of calomel (one to three grains) should be given once at first, followed by a saline purgative. Dark-colored motions are brought away, the spirits improve, and the fever abates. But mercury must not be given beyond an occasional purge.

Emetics are also to be avoided. They increase depression, and are apt to produce irritability of the stomach difficult to subdue.

In the epidemic of 1847 Dr. Peacock found bloodletting of little use, except in the very early stage. It increased prostration, without benefiting in any commensurate degree the pulmonary disease. Leeches, however, were sometimes useful, and counter-irritation of various kinds. During convalescence *sulphate of zinc* was found to be a useful tonic when the expectoration was thin and spumous, and alkalies more useful when it was viscid and glairy (PEACOCK). *Nitrate of potash*, highly diluted and mixed with lemon-juice and sugar, is a most useful drink. From 60 to 120 grains in twenty-four hours may be taken.

In pneumonia it has been found that although a few persons bore the loss of a considerable amount of blood, yet, in general, blood taken beyond a very limited quantity either did not relieve the complaint, or the practice was actually prejudicial. It is in this form of pneumonia that large doses of the *tartrate of antimony* have been found so advantageous. Indeed, it seems distinctly proved that this form of pneumonia will not bear that powerful antiphlogistic treatment which is necessary when it arises from general causes, and is of a more sthenic character.

When the patient was affected with angina, it yielded readily to small local bleedings when the tonsils were swollen, and to small quantities of wine when the tonsils presented little or no increase of size. The derangement of the bowels also readily yielded to purgative medicines when constipated, and when affected by diarrhoea, and accompanied by pain, to mild purgatives and opiates, or to the compound powder of chalk with opium.

But opium must be used with great caution in severe pulmonary complications. Its use ought to be put off till the later stages, otherwise it may increase the tightness of the chest and the dyspnoea. It must not be given till all danger of lung congestion is past. When the fever and other immediately alarming symptoms of the influenza had ceased, there frequently remained a teasing cough, and the convalescents in general complained of languor, want of appetite, and that their sleep was broken and unrefreshing. For removing these complaints, change of air and riding on horseback were most effectual, and to some they were absolutely necessary; and, in addition to these, mild tonics, or the natural chalybeate waters drank at the spas, were of singular service. In slight cases it was sufficient to limit the patient to white fish and puddings, and in the more severe forms to slops and light puddings. The night air was universally prejudicial. It does not appear that any precautionary treatment was of service in preventing the spread of this disease among the attendants on the sick.

WHOOPING-COUGH.

LATIN, *Pertussis*; FRENCH, *Coqueluche*; GERMAN, *Keuchhusten*—Syn., *Stickhusten*; ITALIAN, *Tosse convulsa*—Syn., *T. canina*.

Definition.—*An infectious and (sometimes epidemic) specific disease, preceded and accompanied by fever of variable intensity; attended in the first instance by catarrh, and subsequently by paroxysmal fits of coughing, which occur in numerous short, rapid, spasmodic, convulsive movements of expiration, suddenly followed by a prolonged inspiration, marked by a characteristic sound of a sonorous kind, and variously named the "kink," "hoop," or "whoop." These paroxysms of expiratory and respiratory convulsive movements alternately recur several times, till the fit ends by a quantity of mucus being brought up from the lungs, or till the contents of the stomach are evacuated.*

Pathology and Morbid Anatomy.—The theory of this disease is that a specific morbid poison produces slight primary fever, which for the most part subsides on specific or secondary actions being established. These are catarrh, followed by a peculiar cough and vomiting, ascribed to irritation of the *vagus nerve* by the specific poison. The disease is a "specific pulmonary catarrh;" but very different opinions have at various periods been entertained as to its nature. Its origin appears to have been comparatively of no very distant date, Sprengel not having been able to trace it beyond 1510, when it was endemic in Paris; but its epidemic character was not determined till 1580.

Like other diseases of this class, it appears, as a rule, but once during life, and attacks chiefly infants and children. Dr. Watson gives an instance of a child born with whooping-cough. There are instances, however, of its occurring not only late in life, but also a second time (HEBERDEN). Blache gives a remarkable instance of a grandfather and grandmother catching whooping-cough a second time from their grandchild, all of them laboring under the disease together. Some consider the disease to be a specific affection of the nervous system; others, that it is a catarrh; but both these pathological conditions coexist in whooping-cough. Inflammation does not necessarily accompany the disease, although a state of the mucous membrane exists by which it is morbidly irritable, or susceptible to impressions.

Pathologists have also ascribed the complaint to a morbid condition of the pneumogastric nerve—an explanation supposed by some to be confirmed by the circumstance that that pair of nerves is sometimes found red, with the medullary matter altered in color, dense in texture, and of cartilaginous firmness (KILIAN, AUTENRIETH). Others believe that a specific poison acts on some part of this nerve (TODD).

The results of nineteen post-mortem observations made by Dr. Graily Hewitt during a recent epidemic of this disease (1855), in children varying from one month old to four years, showed the chief lesion to be collapse of the lung-substance—a condition also

known under the various names of *fatal condition*, *carnification*, and *atelectasis*. The experimental test to detect the presence of this morbid collapse is that suggested by MM. Bailly and Legendre, and consists in inflating the lung, the effect of which is to produce uniform distension in a simply collapsed lung; but the force necessary to distend the carnified parts is more considerable, and some portions are not capable of inflation by any force. The air-cells most distant from the roots of the lungs were most liable to this change, and the margins of the lungs were chiefly affected; and there is generally emphysematous distension of the air-vesicles adjoining the collapsed portions of these organs.

Other pathologists have ascribed the disease to cerebral irritation (WEBSTER in *Medical Gazette*). But facts tend to show that the cerebral symptoms are effects, rather than the cause of the disease. In short, the formation and development of *whooping-cough* seem to follow as the result of a specific poison of an unknown kind, but which is communicated through the atmosphere, and seems to affect directly the nervous system and the pulmonary mucous membrane, like *influenza* and *measles*, and, like them, the disease sometimes becomes epidemic.

Symptoms.—It is observed that, in the majority of cases, whooping-cough commences like a simple catarrh, alike in children and adults; afterwards, however, the nervous element of the disease predominates and is combined with the catarrhal element. In the first instance high fever, and the secretion of a viscid mucus from the bronchial mucous membrane, with repeated paroxysms of coughing—several times in a minute—and continuing for many days, or from one to two weeks, are sufficient to indicate a specific catarrh, as distinguished from an ordinary cold. In some cases, again, the nervous element of the disease is mostly developed; so that from the very commencement a kind of *hiccough* exists, or “spasms in the throat,” from the efforts to inspire, causing a whistling through the larynx. The paroxysms of coughing are thus more frequent, obstinate, and severe in whooping-cough than is usually the case in a common cold; and the patient has also a more troublesome sensation of tickling in the throat and inside the trachea—the commencement of the nervous element of the disease—which eventually becomes so characteristic. The catarrhal symptoms may last for several days—three to fourteen,—or even for several weeks—one to four (WILLAN, TROUSSEAU); six to eight (LOMBARD)—before the specific convulsive cough supervenes; and in some instances, where all the other symptoms are present, the convulsive cough may be absent. The fever of invasion is also characteristic of whooping-cough. It is of greater intensity and of longer duration than the fever of an ordinary catarrh. It may last from seven to fourteen days; while the fever of a simple catarrh is rarely prolonged beyond two or three days. In the first stage of the disease it is the incessant repetition of the cough which is most characteristic; in the succeeding stage, when the cough becomes convulsive, the incessant repetition subsides, the cough recurs less frequently, and is more convulsive. A sensation of tickling or prickling in the larynx and

trachea is the indication of a convulsive cough coming on; and no doubt this is the warning which young children recognize and dread as the harbinger of a paroxysm, which suggests to them the necessity of seizing something for support during the fit of coughing, which almost immediately commences. The irritation is attempted to be got rid of by coughing; and in the expiratory efforts the air is expelled with great violence, and so repeatedly and irresistibly that the lungs are ultimately almost emptied of air. At the conclusion of these expiratory efforts the condition of the lungs resembles that produced by *asphyxia*. A sudden inspiration now necessarily and suddenly follows, the air being drawn through the glottis by the gasping patient, with a force and velocity which gives rise to a shrill, sonorous sound, not unlike the crowing of a cock, and which has been variously named a *kink*, a *hoop*, or *whoop*; and the disease has accordingly received various names, such as *kink-host*, *hooping-cough*, *whooping-cough*, *chincough*. The anxious and distressing inspirations are scarcely completed when the convulsive expirations of the cough are again renewed, and again followed by the gasping and crowing inspirations, till a quantity of mucus is brought up from the lungs, or till the contents of the stomach are rejected by vomiting. Such are the phenomena of the fit or paroxysm of whooping-cough. After it is over, the patient in ordinary cases appears to be but little affected, and returns immediately to play, or to any other occupation which takes the attention at the time.

When these phenomena are prolonged, secondary effects are produced, whose morbid appearances have been noticed. The immediate consequence of the violent fits of coughing is to interrupt the free transmission of blood through the lungs, and the return of blood from the vessels of the head. This causes not only the turgidity, swellings, redness, and lividity of the face and eyelids which attend the fits, but also the discharges even of blood from the mucous surfaces of the nose, ears, or eyes. The little sufferer may shed tears of blood.

Whooping-cough varies greatly in intensity, and most authors divide the group of symptoms into three stages. The first stage comprehends the period from the first symptoms of illness until the "*whoop*" confirms the convulsive nature of the cough. This is the period of development or evolution. The second stage commences as soon as the nature of the cough is determined, and lasts till the violence of the cough and the danger of secondary complications is past. This is the period of spasmodic paroxysms characteristic of the disease. The third stage comprehends the convalescence of the patient, until the final and happy termination of the disease; or the occurrence of any event which may destroy the sufferer.

When the convulsive stage of the disease is fully formed, and the series of fits or paroxysms of severe coughing occur at uncertain periods, during the interval the patient generally enjoys his usual health, recovers all his gayety, returns to his play, and relishes his food with a good appetite.

A paroxysm or fit of whooping-cough comprehends the following phenomena:

The approach of the fit is notified to the patient by an unpleasant titillation of the glottis, by a sharp pain in the chest, or by a spasmodic contraction of the diaphragm. As soon as the child is thus warned, he instinctively ceases from play—his spirits suddenly droop, and he runs to his nurse, and either grasps her arms, or lays hold of her chair, or her dress, to support himself during the paroxysm, which in a few minutes or seconds is about to follow. In severe cases the cough is quite convulsive, and so rapid is the action of the diaphragm, that the air is almost instantly expelled from the lungs, and the patient, half-suffocated, turns black in the face, and frequently passes urine. At length the crisis approaches, the diaphragm relaxes, and a violent inspiration follows, accompanied by the characteristic *whoop*. This sound perhaps remits, but after a few seconds returns; and thus convulsive inspirations and expirations continue, till the patient is at length relieved by a copious expectoration, or by vomiting. The matters expectorated from the lungs are frequently thick, ropy, and viscid; or a colorless liquid. When vomited from the stomach, the patient throws up a glairy fluid of much tenacity, semi-transparent, and frequently amounting to the greater part of a pint; and should he have recently eaten, the food returns with it. It frequently happens, however, that the stomach retains the food and rejects the offending matter. If the fit be violent, the fluid rushes not only from the mouth, but also from the nostrils; and in some instances is mixed with blood, for blood occasionally bursts forth in considerable quantities from the congested vessels of the mouth, the nostrils, the ears, the eyes, and in some instances from the rectum. Such cases are most severe.

If the stethoscope be applied to the chest previous to the fit, the mucous rhonchus common to catarrh may exist, yet in most cases the respiration is natural. During the act of coughing the respiration appears completely suspended, and is not sensible to the ear in any part of the chest. On the "*whoop*," however, taking place, the air is heard to rush with remarkable violence into the trachea; but at this point it stops for one or more seconds till the bronchial tubes relax, and the air is then admitted into the lungs.

The fit having subsided, the eyes, which seem to have started from their orbits, resume their natural position, but are inundated with tears, or the conjunctiva is more or less gorged with blood; the natural expression and appearance of the countenance returns, and in a few minutes, in favorable cases, the good spirits of the little patient are renewed, and he eats with appetite. On the contrary, in severe or unfavorable cases, long-continued exhaustion, headache, and some fever, are the preludes to convulsions, inflammation, or the other more severe complications of the disease.

The paroxysm varies greatly in frequency and severity, but, in general, its frequency is as its severity. In ordinary cases it returns every two hours; but in severer cases, and especially during the second and third week, it returns every half or every quarter of an hour, or even oftener. This disease commonly reaches its acmé at the end of the third, fourth, or fifth week; after which the paroxysms diminish in frequency, the intervals are prolonged, and the patient

is to a certain degree convalescent. The duration of this second stage is from two to six or eight weeks.

The third stage commences with the convalescence of the patient, when the paroxysms become milder, the intervals longer, the expectoration thicker and more opaque, greenish, or pus-like, and more like ordinary catarrh. The vomiting ceases, and the general health of the patient begins to improve greatly. The duration of this stage however, is often long and variable, and the cough may still harass the patient for many weeks, or even many months. It is to this stage that the term chronic is usually applied.

The disease lasts from six to eight weeks; but there are exceptional cases, which on the one hand get well in a week, and on the other hand may continue several months, or even a year. "The general duration of the disease is directly proportionate to the duration of the prodromata;" and the more quickly the convulsive cough makes its appearance, the more quickly does the disease subside (TROUSSEAU).

Many accidents may arise to complicate the symptoms of whooping-cough, and to increase the danger, as inflammation of some of the tissues of the lungs, of the mucous membrane, of the stomach or intestines, or of the serous membranes of the brain.

Inflammation of the minute bronchia is the most usual complication of this disease—*capillary bronchitis*, or *peripneumonic catarrh*. The form of inflammation may be that in which the secretions are in defect, so that the mucus is not only greatly diminished in quantity, but is thick and viscid, teasing the patient with fruitless efforts to free it from the air-tubes, and thus causing a frequent recurrence of the paroxysm. In other cases it may assume the form of purulent inflammation, the pus secreted being formed into sputa, and moderate in quantity; or it may be thrown up pure, as from an abscess, and so enormous in quantity as to amount to one or two pints in the twenty-four hours. The inflammation of the bronchial membrane may spread to the substance of the lungs, when the danger, as well as the symptoms, of some of the various forms of pneumonia will be added to the disease; but the most formidable accident is when the pleura is inflamed, for then the patient's sufferings during the paroxysm are fearfully increased, from the agonizing pain inflicted during the paroxysm of the cough. These lesions are the most frequent causes of death in cases of whooping-cough.

The mucous membrane of the stomach and intestines is often the seat of inflammation; and this is denoted by pain in the epigastrium, and by the suppression of the glairy fluid thrown up by vomiting, so that on the termination of the fit the patient often lies in a state of complete exhaustion, unable to discharge anything either from the stomach or lungs, or even to "*whoop*;" and he is then said, in popular language, to labor under the "*dumb-kink*."

In mild cases the bowels are little affected in this disease, except that the patient sometimes passes his feces during the paroxysm. In severe forms the stools are often either black and offensive, or they consist of a colorless mucus, the latter evidently depending on an inflamed state of the mucous follicles.

Headache is a symptom which usually attends the catarrhal stage, but generally ceases when the fever subsides. In some instances it continues throughout the disease, and is not unfrequently the forerunner of fatal convulsions, or epilepsy, or of inflammation of the membranes of the brain, terminating in delirium, coma, hydrocephalus, and death.

Diagnosis.—It is impossible to determine whether the febricula of the first stage is the result of simple catarrh, or will, on its subsiding, prove to be whooping-cough. The earliest recognition of whooping-cough is by the *obstinate coughing*. A cold giving rise to paroxysms of coughing recurring fifteen, twenty, or thirty times in a minute; and which continues in this manner for four to ten days in succession, attended with high fever, is certainly a specific catarrh; and after a period varying from one to two weeks later whooping-cough will manifest itself with its well-marked characteristics (Trousseau). As soon, however, as the cough has been followed for two or three paroxysms by the "*whoop*," the diagnosis is perfect, no other disease being accompanied by this symptom.

Cause and Modes of Propagation.—That whooping-cough is induced by a specific poison there is little doubt; but in what manner this agent is generated is not determined. The disease is always sporadic, sometimes epidemic, and the "epidemic influence" is the most common cause and mode of propagation.

The predisposition to the disease is so strong that few persons pass the period of childhood without suffering from it; but it may occur at any subsequent age. The early age at which the large majority of patients pass through the disease is, however, a sufficient reason for our very slight acquaintance with the predisposing causes.

When whooping-cough is once excited, the patient evolves a poison which is both infectious and highly contagious, communicable from one human being to another—a fact which necessarily implies the idea of "specificity." The general public are so unanimously of opinion that this disease is infectious, that no parent will permit his yet unaffected child to mingle with such as may be laboring under it. The profession are, with a few exceptions, of a similar opinion; and it is probably most contagious at the period of its highest development.

The infecting *distance* of the poison must be considerable, from the utter impossibility of isolating the patient at home, or of preventing the spread of the disease in schools.

Rosen conceives that, without being aware of it, he has often carried the disease from house to house. Frank also says that it is often propagated from patient to patient, from house to house, and from village to village. In Geneva, Lombard has often traced the first cases occurring in that city to a neighboring town, or to a sick child from the country. Whooping-cough was some years ago introduced into St. Helena, where it proved very fatal: the captain of a ship, having some children laboring under the disease on board, allowed their dirty linen to be sent on shore to be washed, and so introduced the disease among the inhabitants.

The poison of this disease may coexist with many other poisons, and in this case they often greatly influence each other's actions. *Small-pox* and *whooping-cough* have often coexisted; and a very common and fatal combination is *measles* and *whooping-cough*. *Whooping-cough* and *cow-pox* are not unfrequently combined. Indeed, the lower classes erroneously look upon vaccination as in many instances a cure for *whooping-cough*.

Period of Latency.—The disease has a stage or period of incubation, but our knowledge of the extent of this period is at present extremely imperfect. The disease never shows itself immediately after exposure to contagion, but a certain number of days (five or six) elapses before the symptoms of catarrh are to be observed.

Prognosis.—The proportionate number of deaths to recoveries in *whooping-cough* is not determined, but greatly varies in different years; for in one year hardly a death will occur from the disease in a large city, while in another year many children will die. In general, however, the milder forms of the disease are rarely fatal, while the more severe and protracted cases very commonly are so. Lombard thinks station in society greatly affects the mortality; for he says that of ten fatal cases nine belong to the poorer classes. The reports of the Registrar-General show that the mortality is greater from this disease in towns than in the country, being in the metropolis, in 1838, .111 per cent., while in England and Wales it was .061. In the year 1839, also, it was for the metropolis .061 per cent., while for England and Wales it was .053. Lombard gives the ages of forty fatal cases as follows:

AGES.	CASES.	AGES.	CASES.
From birth to 6 months, . . .	6	From 4 to 5 years,	2
" 6 to 12 months, . . .	7	" 5 to 6 years,	2
" 1 to 2 years, . . .	10	Above 6 years,	0
" 2 to 3 years, . . .	6		
" 3 to 4 years, . . .	7	Total,	40

Danger from bronchial inflammation is to be dreaded rather towards the end than the beginning of the disease. Convulsions are apt to occur if dentition is going on at the time; and if they arise from the congestion or effusion within the cranium, the case is generally fatal. The number of paroxysms which a child may have in twenty-four hours is the best basis of prognosis. Twenty fits in twenty-four hours denote a very mild case; when more violent, forty to fifty paroxysms may occur in that period; and when the number of paroxysms exceeds forty, the case is a serious one, and prognosis grave.

If a predisposition to tubercle exists, *whooping-cough* may determine the development of *phthisis*.

Treatment.—On the invasion of the disease, beyond putting the patient on a low or very moderate diet, and attending to the daily action of the bowels, there is little occasion for medicine.

The "*whoop*" having confirmed the nature of the affection, and the second stage being established, the disease will run its course, and one of two indications of treatment may be followed. The

first is to prevent, if possible, convulsions, or any attack of inflammation, either of the lungs, the stomach, or of the membranes of the brain. The second indication, after the period of danger is past, is to prescribe such medicines as may diminish the frequency of the paroxysms. Indeed, the best mode of obviating the danger of cerebral irritation, or of inflammation of any of the organs that have been mentioned, is to mitigate and control, as far as possible, the frequency of the paroxysms, to check those secretions which are in excess, and to excite those which are in defect, and these objects are best obtained by mild sedatives, combined with gentle purgatives or laxatives.

The choice of the sedative has been considered a matter of much importance. The continental physicians have bestowed much praise on *belladonna*, others on *hemlock*, others on *henbane*, while some have contented themselves with *opium*. It must be admitted, however, that none of these narcotics possess any specific property in controlling this disease, so that the selection of the particular one must be left to the discretion of the practitioner. But supposing the patient to be a child, as the head is especially the organ to be protected in such cases, the mildest sedatives, such as *hyoscyamus*, or the *syrup of poppies*, are the safest and best. Should, however, *belladonna* be selected (as it seems to be the most efficient sedative), if the child be under four years of age, the dose ought not to exceed *one-eighth or one-tenth of a grain* of the extract, with the same quantity of the powder of the leaves in a pill, the pill being dissolved in syrup at the time when it is to be administered (TROUSSEAU). The dose should be given in the morning; and only once daily, commencing with one pill, and increasing to two or more. Or if *hyoscyamus* is chosen, *half a grain to a grain* every six or eight hours; while, if it be the *syrup of poppies*, this medicine should be given in such fractional doses of a *drachm* as are suited to the age. Powdered *belladonna* root has been recently recommended by Vollaut. The dose is *one-fifth of a grain*, given at first *once*, then *twice*, then *four times a day*, and so on until the paroxysms begin to subside, when it is to be given at much longer intervals. Thus, he says, the spasmodic period may be positively arrested in three or four days. The powder of the leaves he considers to have little efficacy (*Syden. Society Year-Book*, 1862). Infusion of wild thyme, slightly sweetened and mixed with gum, is also said to effect great improvement in cases of spasmodic cough (JOSET, *l. c.*). *Cochineal* is an anodyne which sometimes affords relief. It is usually prescribed in the form of a mixture, consisting of *cochineal*, 10 grains; *subcarbonate of potash*, 20 grains; *sugar*, $\frac{1}{4}$ oz.; *water*, 4 oz.; *rub together and strain* (*Squire's Companion to the Pharmacopœia*, p. 62). Of this mixture, a quarter of a teaspoonful four times a day is sufficient for a child one year old; half a teaspoonful for a child of two years; and a teaspoonful for a child of four years. [Or the bromide of potassium, or bromide of ammonium, may be given.] Boiled apples in milk should be given for food.

An opiate, in the early stage of the disease, ought not to be administered alone, and some purgative or laxative ought, as a general

rule in all cases, to be combined with it. The selection of the particular medicine is perhaps unimportant, and any vegetable or saline purgative will perhaps answer equally well, as the *confectio sennæ*, *rhubarb*, *castor oil*, or *manna*. The neutral salts, however, sit easiest on the stomach, and (as the medicine must be continued) are the most agreeable to the patient. *Opium* is a dangerous remedy, and is liable to the objection of being apt to check the mucous secretion.

If at the outset or afterwards the cough is very suffocative, an emetic is useful. Five to nine grains of sulphate of copper dissolved in three ounces of distilled water, and a dessert-spoonful every ten minutes is the most efficient (TROUSSEAU).

Nitric acid, in the following formula, has been found of service:

Acid. Nitrici diluti, fʒxij; Tinct. Cardam. comp., fʒiij; Syrup, fʒiiiss.; Aquæ, fʒj; *misce*. Of this mixture, one or two small teaspoonfuls may be given every two hours (GIBB).

Towards the close of the second stage the symptoms may become unfavorable, and cerebral irritation, with convulsions, or inflammation of the membranes of the brain, of its substance, or of the tissues of the lung, or of the alimentary canal, may complicate the disease, and then the treatment of the case is always exceedingly difficult, and frequently unsuccessful.

If convulsions should come on suddenly, and without headache or other symptom of inflammatory action, small doses of any opiate, and *mustard poultices* to the feet, may relieve the patient; but should convulsions still continue, an *asafoetida injection* may be administered. It often happens that convulsions are combined with a suppression of the vomiting, and of the usual glairy discharge; and in these cases *leeches*, followed by a large *linseed poultice*, should be applied to the epigastrium. If the unfavorable symptoms should advance, and headache or other symptom show an affection of the membranes of the brain, *leeches* should be applied to the temples and *cold* to the head.

When the poison excites inflammation of the tissues or substance of the lungs, *bleeding* to a limited amount by leeches may be required; but we should be satisfied with such mitigation of the symptoms as may obviate immediate danger, and even that is not always obtained, since the affection is not to be subdued by bleeding, as in simple inflammation; for, being dependent on the action of a morbid poison, it will run a definite course. Blache bled in nine cases, either with the lancet, by leeches, or by cupping, and in one case no less than five times; yet, he adds, with a desolating want of success, and eight out of the nine cases terminated fatally. This result makes him add an axiom, in which every practitioner will agree, that there is in severe *whooping-cough*, as in *typhus*, *cholera*, and many other affections, an unknown element which modifies and gives a specific character to all these intercurrent inflammations.

If the intestinal canal be affected, some purgative, combined perhaps with *calomel*, may be necessary to act on the bowels and free them from their contents; and if the stools be white and muciform, and the patient not relieved, an enlarged state of the follicles may

be suspected, and consequently a *linseed poultice* should cover the abdomen for some hours, preceded, perhaps, by an *enema of syrup of poppies and barley-water*, and which should be administered night and morning. Many other modes of treatment have been recommended for the cure of whooping-cough, and more especially by *emetics* repeated every second day. For obvious reasons such a method is not to be recommended.

The disease having passed into the third stage, and the inflammation or other threatening symptom, if any has existed, having subsided, it is desirable to attempt to abridge the duration of the cough, which often extends to a most distressing length; and for this purpose *tonics*, *antispasmodics*, *counter-irritants*, and other remedies, either externally or internally, have been recommended.

The more stimulant antispasmodics, as *asafoetida*, *musk*, *castor*, *oil of amber*, *cantharides*, and *camphor*, are the remedies which have obtained the most suffrages in the cure of this stage of whooping-cough. The two first are in most esteem; and some persons even considered *asafoetida* to be a specific, not only in this, but in every other stage of the disease. It should be given in emulsion, in the dose of one or two grains to a child two years old, repeated three or four times a day, or even as often as every two or three hours. Cullen, however, preferred *cinchona* to *asafoetida*, and considered it "the most certain means of curing the disease." Many other remedies have been mentioned, as *alum*, *hydrocyanic acid*, *oxide of zinc*, *arsenic*, and many preparations of *iron*, and all of these remedies have been found to a certain extent useful as tonics; but in estimating the results of remedies, we should be careful not to mistake temporary recovery for cure; and the fact of so very many remedies being highly spoken of suggests doubt as to the value of either.

When internal remedies have failed to make any impression, the cure is often attempted by means of local treatment, or by derivatives. The early physicians applied *actual cautery* to the nape of the neck; the modern ones, *blisters* to the spine, or directed the back to be rubbed with the *unguentum antimonii cum potassio tartarizati*, or with some liniment or embrocation, as the liniment of *camphor* or of *ammonia*, or with *asafoetida*, *oil of amber*, *oil of turpentine*, or the *tincture of cantharides*. The general opinion, however, is, that these do little good unless they contain some opiate, whose absorption they facilitate, and this is attended with danger. Foot baths and the warm bath have also been used, and often with much efficacy.

When ordinary remedies have failed, a change of air is a resource of great value, and was first mentioned by Dr. Forbes, in his thesis *De Tussi Convulsivi*, in 1754; and since that period it has been recommended in dangerous cases by most physicians with that praise it so eminently deserves. While it is determined that a change from the bad air of a town to the purer air of the country is at all times a great benefit, Lombard contends that he has found a change from the country to the town to be not less beneficial, and that the patient is benefited even by the removal to so short a distance as half a mile. Indeed, it is impossible to witness more striking instances of the advantages of treatment than we occasionally observe in pa-

tients when removed from large towns to their environs, for even in a few hours they have been known to recover from an apparently hopeless state. A sail across a river is also beneficial, although the distance may be short.

Dietetic and General Treatment.—The patient should not be allowed animal food from the commencement almost to the termination of the disease *in its acute form*. It is desirable also that the temperature of his apartment should be regulated, and that he should not be exposed to any considerable or sudden change from heat to cold. In mild weather also, if no local symptom forbids, he should be permitted to take exercise in the open air. He should likewise wear flannel.

There are no known means of prevention, except an entire removal from every source of contagion.

MUMPS.

LATIN, *Parotides*; FRENCH, *Oreillon*; GERMAN, *Ziegenpeter*—Syn., *Mumps*; ITALIAN, *Parotitide*.

Definition.—*An inflammation of the parotid and salivary glands, probably specific, and certainly in some cases contagious and epidemic.*

Pathology.—It is most common in male children; and is less frequent after puberty; and second attacks of the disease are rare. The disease is sometimes epidemic, and in certain localities it prevails rather than in others; so that local causes may have to do with its propagation and maintenance.

The disease often occurs also in the course of severe fevers (typhus and enteric); and it has been noticed in cases of cholera. It is a result also of ptialism from iodine or from mercury. Dr. John Harley gives the following example of its direct propagation from person to person. "A medical student had *mumps* in London, at a time when his mother was staying with him. They remained in town till the swelling disappeared, and then went—a hundred miles into the country—home. There was no *mumps* in that neighborhood; but a fortnight after their arrival one of the children was taken with the disease, and it afterwards successively affected, at regular intervals of a fortnight, each member of a large family" (Hooper's *Physician's Vade Mecum*, 7th edition, p. 558).

Symptoms.—Febrile phenomena, associated with pain and uneasiness in the region of the parotid. The pain on moving the jaw soon becomes so great that mastication becomes impossible in severe cases. Considerable fulness and soreness prevail at the angle of the jaw over the malar region and region of the parotid, on both sides generally. Beneath one or both ears redness prevails, with pain on pressure; and the pain becomes so great as to prevent sleep at night in severe cases; and in such cases the swelling generally extends to the submaxillary glands, and to the tonsils, and neighboring parts of the pharynx, so that swallowing becomes very difficult and painful. The region of the swollen glands becomes tense and glossy; sometimes of dusky livid hue; and when both sides

are affected at the same time, the face is of an enormous size; and continues so for four or five days, after which it gradually subsides, and resolution ensues, the fever and pain gradually subsiding. It is rare that the inflammation ends in suppuration. Occasionally during the course of the disease, but generally towards its subsidence, the testicles swell, or the mammae in the female; and in some cases the cerebral membranes become implicated—as also the gastro-enteric mucous membrane.

Treatment.—Constant hot fomentations (after leeching in severe cases) should be applied to the swollen parts. The maintenance of a constant but gentle action on the bowels, by saline cathartics, must be attended to. Absolute rest ought to be enforced, and a farinaceous diet enjoined. The disease runs a definite course, and is not likely to terminate unfavorably.

DIPHTHERIA.

LATIN, *Diphtheria*; FRENCH, *Diphthérie*; GERMAN, *Diphtherische Entzündung der Rachenschleimhaut*—Syn., *Diphtheritis*; ITALIAN, *Difterite*.

Definition.—*An acute specific general disease which runs a quick and definite course in eight to fourteen days. Its anatomical character is spreading inflammation of the mucous membrane of the pharynx, attended by exudation of lymph, also by swelling of the submaxillary and cervical glands, and of the spleen. The disease is attended with great prostration of the vital powers; by a very early appearance of albumen in the urine, which may continue for a very short time only, or may become persistent. In some cases a remarkable series of nervous phenomena are apt to supervene, characterized by progressive paralysis, and sometimes by fatal syncope. A membranous exudation similar to that which appears on a mucous surface occasionally also appears on a wound.*

Historical Notice.—Diphtheria is by no means new to England; and the writings of the older physicians prove that from time to time it has been epidemic, or at least very common in many parts of England. The historical accounts of the disease show that it has preserved its essential character and nature from age to age. Ever since the end of the “sixteenth century, diphtheria has been observed in every region of the Old and New World.” At first it continued for a time in Spain; and during nearly forty years was noticed in different parts of the Peninsula. Rather later, all Italy was successively afflicted by it. Towards the middle of the last century especially, epidemics of the disease have occurred, less general and less prolonged, but more multiplied, in England, in France, in Sweden, and in America, and particularly at New York and Philadelphia. It terminated the life of Washington, and of the Empress Josephine. The outbreaks have usually been limited in extent, sometimes not spreading beyond a single dwelling, building, village, or quarter of a large town. It prevailed as an epidemic in the north of France and south of England in 1859, to a con-

siderable extent; and since that time many excellent monographs have been written on the subject in our own country, among which those of Hunter Semple, Chatto, Wade, Ernest Hart, Greenhow, Sanderson, and Jenner are conspicuous.

Pathology and Morbid Anatomy.—In this disease, as in many others of the *miasmatic* kind, the *general* or the *local* symptoms may predominate, giving special features to each case; and the patient may die from the severity of the general disease, or from the severity of some one of the local lesions.

The mucous membrane covering a tonsil may be the primary seat of the characteristic local exudation, or the arches of the palate, or the posterior surface of the soft palate, the uvula, the nares, or the pharynx may be the primary seat. At first there is redness and swelling; and the normal mucous secretion is so altered in its physical properties that it adheres by its own increased viscosity to the mucous membrane. A white or gray patch now forms on the membrane, which indicates the presence of a layer of lymph on the reddened surface.

The layer of lymph may thus spread from one or from several centres over the reddened surface; and this redness may involve the whole mucous membrane within reach of the eye. The lymph which grows upon this reddened surface may descend into the larynx, the trachea, and the bronchi. Dr. Stokes has recorded a fatal case, in which the tongue, tonsils, pharynx, epiglottis, larynx, trachea, and right bronchus were more or less thickly coated with the deposit, even as far as the fourth or fifth bronchial ramification, while the left bronchus remained quite free from it. The right lung was œdematous and consolidated, the left comparatively healthy (*Dub. Jour. Med.*, Feb., 1863). Dr. Jenner has known the diphtheritic exudation to extend into the œsophagus and stomach (*Diphtheria; its Symptoms and Treatment*, by Dr. Jenner, p. 4). If the lymph be torn from the mucous membrane, a raw, bleeding surface is exposed, which in a few hours is again covered by a new layer of lymph. The lymph of diphtheria has a variety of appearances. Sometimes it is granular, with very little consistence or tenacity. Sometimes the part is covered with a pulpy substance of a white or gray color; but this pellicle is constant in some form or other, and is possessed of the power of reproducing itself. It is this specific exudation which establishes the disease as one *sui generis*, and to which Bretonneau gave the name of "*Diphtheritis*," and which has been subsequently modified to "*Diphtheria*." The latter term has the advantage of being the shorter word, and is that adopted by the Registrar-General. Etymologically, the terms are derived from *διφθέρω* *vel* *διφθερίω*, signifying the prepared skin of an animal; while *διφθερίτης* *vel* *διφθερίτης* signifies that which is covered with a fur, or with a leathern coat.

In microscopical characters it does not appear that this "fur," "pellicle," or "false membrane" of diphtheria can be distinguished from the concrete exudation on blistered surfaces, or that which forms in the angina of scarlatina (*EMPIS*). The commencement of the formation of the pellicle is in reality an act of coagulation. The

mucous membrane exudes, in the first instance, a fluid in which the *fibrine* or *mucin* coagulates; and such coagulated material forms the tube-casts which line the surface of the larynx and trachea, but from the mucous surface of which they come to be separated by a considerable interval; and generally it may be stated that there is the greatest possible variation as to the extent, the consistence, the color, and adherence of the pellicle. Sometimes the particles of lymph are so thin, soft, and separated from each other, that the term membrane can scarcely be correctly applied to it. At other times it is tough, elastic, and as much as an eighth of an inch in thickness. In the one case the lymph resembles cream in consistence; in the other it resembles wash-leather; and between the two extremes we meet with all intermediate conditions as regards consistence and tenacity. Pus, granular corpuscles, oleo-protein granules and epithelium constitute the bulk of the softer forms of the so-called lymph; while such fibres as we see in the buffy coat of blood-coagula constitute the bulk of the toughest variety of the lymph pellicle (JENNER). Vegetable growths, as the *oidium of muguet* (VOGEL), occur in the pellicle of diphtheria from time to time, and have been reported by some as a constant occurrence. It is, however, by no means so; and the accidental existence of such vegetable growths is no evidence that epiphytes have any essential connection with cases of diphtheria (JENNER).

The lymphatic glands to which the lymphatics of the pharynx lead, are found in cases of diphtheria to be larger, redder, and moister than natural; and if the disease has continued long, they become brittle, pale, and of a brightish-red color on section—characteristic of inflammation of their substance. These enlarged glands may be felt during life behind the angle of the lower jaw on either side, as well as down the neck by the sides of the larynx, when that organ is implicated. Such enlargement of the glands is just in proportion to the severity and depth of the local, nasal, pharyngeal, laryngeal, and tracheal disease; and when the discharges from the pharynx are fetid, and the mucous membrane sloughy, not only are the glands behind the angles of the jaw enlarged, but the connective tissue in which they are placed is the seat of the effusion of serum, and even the exudation of lymph, so that very great general swelling of the parts is the result.

Condition of the Urine in Diphtheria.—Albumen is found in many cases; 50 per cent. (LEE), 66 per cent. (BONCHUT, EMPIS), in the majority (MANGIN), in all the cases examined by the observer (SANDERSON). Its quantity appears to be sometimes enormous, so that the urine becomes quite solid from heat and nitric acid (PARKES, *l. c.*).

Although it is not established that albuminuria is an essential element in the disease, yet it is a most important symptom, both as connected with the pathology of the disease and with its prognosis. The first discovery of the relation of albuminuria to diphtheria was made by Mr. Wade, of Birmingham, and was communicated to the Queen's College Medico-Chirurgical Society in December, 1857. During the following year Bonchut and Empis made a similar discovery in Paris. These observers attach very great importance to

the renal complication, as affording an anatomical explanation of the fact, that, in many cases of diphtheria in which death occurs neither by suffocation nor by septic poisoning, it cannot be due to local lesion. Bonchut considers it a sign of the commencement of purulent infection in diphtheria, and coincides with very great gravity of the disease. The blood then assumes the tinge of bistre; and numerous masses of pulmonary apoplexy may be found after death, resembling those which precede the development of metastatic abscesses in the lungs. Mr. Wade says (*The Lancet*, 1862):

"When my attention was first turned to diphtheria, the doctrine of Bretonneau was paramount, that diphtheria is a local disease, and for the most part always remains so, infecting the system, if at all, only by absorption of putrescent matters from the throat, and, consequently, all treatment was to be local. I was struck early by the fact that many slight cases die; and I resolved to make a full dissection as soon as opportunity should offer, without any preconception as to what I should find. In the first case I found (after about seven days' illness) a pair of white kidneys, such as one finds after scarlatinal dropsy. The spleen was occupied by a similar deposit. After this I turned my attention to the kidneys during life, and found not infrequently *albuminuria*. I have never said (because I never believed) that *albuminuria* and *uræmia* were convertible terms; but I do say that I have seen many cases of diphtheria which presented distinct symptoms of *uræmia*—comatose or comatoid phenomena—coincidentally with an obvious diminution of the urinary secretion, and relieved (and that very suddenly) by restoration of the secretion. I looked upon the discovery of renal complication as important, for two reasons,—*Firstly*, because it showed that diphtheria does not spread solely (as Bretonneau taught) by continuity of surface. *Secondly*, as accounting for death in *certain* cases previously inexplicable.

"From considerations flowing mainly from the discovery that the 'continuity of surface' theory was erroneous, I revived the doctrine that diphtheria was an essential fever, and hence the inutility of local treatment, at that time universally carried out, under the influence, and as a corollary to, the theory of its being a local disease.

"In Dr. Sanderson's essay, he seems to suggest that I had overlooked the fact of there being hyperoxidation in diphtheria. So far from ignoring it, it formed the basis of my views.

"Dr. Sanderson relates a case in which there was, with *albuminuria*, abnormal amount of renal excretion, and says that this case proves that my views are incorrect, and that in no case does insufficient elimination exist. I suggest that, *in the first place* (admitting all the facts of this case to be as he has stated), to draw the inference that, in no case can there be insufficient elimination, is to commit the logical error of reasoning from a particular to a universal. *In the second place*, admitting that the renal elimination going on during the height of the pyrexia was vastly greater than that of a period of health or convalescence when the ingesta were greater, proves nothing at all as to the adequacy of the elimination during the former period. The real question is this—'*Was the elimination adequate to remove from the system the abnormally excessive quantity of effete material produced by the pyrexia?*' To this Dr. Sanderson's comparative experiments give no answer. There are no scientific data in existence which can answer it. I admit that it is eminently desirable that these should be procured. But this is to be done, not by comparing

the total daily excreta during convalescence with those of a part of the pyrexial period, but by comparing the excreta of the 'pre-renal' pyrexial period with those of the 'post-renal.' In the meantime, the answer I offer to the question is based upon clinical experience. I find a certain amount of urine, of a certain specific gravity, *without* certain general symptoms one day, and the next day I find urine of less specific gravity, probably less in total quantity, *with* certain general symptoms. These symptoms resembling some of those found in cases of pure kidney disease (and commonly reputed to depend upon that disease), I infer that, in these cases also, the symptoms depend upon insufficient elimination. I do not say that we find these symptoms in all cases of diphtheria. I do not say even that we find them in all cases of diphtheria with albuminuria.

"As regards treatment, I recommend the adoption of the Cullenian doctrine here, as in other specific pyrexiae—namely, 'to avoid the tendency to death.' One tendency is to death by insufficient elimination of effete material. I try to avoid this *principally* by copious supplies of aqueous liquids. I do not object to meet other tendencies by appropriate remedies, whether of a stimulant, tonic, or any other suitable character.

"I also strongly condemn the indiscriminate employment of topical applications, as being most painful and distressing to the patient, and commonly inoperative. On the other hand, some of the most striking recoveries I have seen have been unquestionably due to the timely use of local means suitable to the exigencies of the particular case."

Dr. Sanderson is of opinion that neither of these doctrines regarding the pathology of albuminuria in diphtheria is the true one. In several of the cases related by him the cessation of albuminuria was coincident with amelioration of the patient's condition and the disappearance of the most alarming symptoms. The early period of the disease at which the albumen appears, and the short time during which it lasts, are facts of great importance. Dr. Sanderson is of opinion that either (1), the kidneys must be the seat of the primary morbid process; or, (2), the albuminuria must depend on an original change in the blood. That it is not due to the former of these is evident from the fact that the renal disease is only coincident with disease elsewhere (*e.g.*, in the fauces), so that the special morbid condition of the blood induced by the *diphtheria miasm* must be regarded not only as the primary cause of albuminuria, but of all the other symptoms. This Dr. Sanderson illustrates by comparing the poison of diphtheria to that of the poison of cantharides, which, from the moment it enters the circulation manifests its presence by albuminuria, and produces a series of anatomical changes in the kidney, which are identical with those described by Mr. Simon and Dr. Bristowe in diphtheria. Dr. Sanderson's observations still further show that, at the acmé of the disease, when the urine was intensely albuminous—when there was complete anorexia, and the ingesta reduced to a minimum—that then the quantity of urea excreted in twenty-four hours was about twice as great as that excreted during a similar period when convalescence was established—when the patient was eating, with an appetite, the ordinary hospital diet, with extras. Thus it is shown that diphtheria agrees with the other pyrexiae in being attended with a marked increase

in the excretion of urea, and that the existence in the kidney of the condition implied by albumen and fibrinous casts in the urine, does not necessarily interfere with increase in the elimination of nitrogenous material. There is, therefore, no reason to apprehend the occurrence of uræmia as a consequence of the renal complication in diphtheria, this complication not being the cause of the blood-poisoning, but merely the index of its existence (*Brit. and For. Med.-Chir. Review*, Jan., 1860, p. 196).

Phenomena and Symptoms.—The *prodromata* which forebode an attack of diphtheria may be set down as general *malaise*, anorexia, slight fever, dysphagia, and glandular swelling. The symptoms generally supervene very gradually and insidiously; but feelings of depression, prostration, and muscular debility prevail, attended by headache, nausea, diarrhœa, and chilliness. There is a sense of stiffness about the neck and throat, and the drowsiness which often attends the accession of an attack of diphtheria may lead the patient to fancy he has caught a slight cold in the throat while indulging in a short sleep.

Dr. Jenner has grouped his cases of diphtheria into *six* varieties, as follows: (1.) *The mild form of diphtheria*; (2.) *The inflammatory form*; (3.) *The insidious form*; (4.) *The nasal form*; (5.) *The primary laryngeal form*; (6.) *The asthenic form*.

In the *mild form of diphtheria* the general symptoms and the local lesions are trifling, and no sequelæ follow. Febrile disturbance prevails to a slight degree; and there may be the least possible soreness of the throat on swallowing. No albumen occurs in the urine, and no nervous symptoms follow. Dr. Jenner is of opinion that many inflamed throats, when *diphtheria* is epidemic, have their origin in the *diphtheria miasm* (whatever that may be), just as many cases of *diarrhœa*, when *cholera* is epidemic, originate in the *cholera miasm*; and it is as difficult to say in some cases that an inflamed pharynx is not due to mild diphtheria as it is to say that a serious diarrhœa is not cholera.

In the *inflammatory form of diphtheria*, symptoms of severe *cynanche pharyngea* precede the exudation of lymph. There is redness, of a vivid or dusky hue, and swelling of the mucous membrane, covering the arches of the palate, the uvula, and the tonsils. The swelling is often considerable, from the effusion of serum into the sub-mucous tissue, which becomes of a jelly-like transparency and aspect. The pain in the act of swallowing is great, so that deglutition becomes impossible. The febrile disturbance may be extreme or moderate; and although the pulse is frequent, it soon becomes weak, and there is the sense of considerable prostration. In from twelve to forty-eight hours after the first symptoms of the throat affection supervene, a layer, more or less extensive, of tough lymph coats the inflamed surface, and death may follow from extension of the exudative process into the larynx or trachea. The urine may contain albumen, and sometimes the joints are swollen, hot, and tender.

The *insidious forms of diphtheria* are dangerous, because they seem sudden and unexpected. The general symptoms are not severe.

There is no marked soreness of the throat, no notable swelling of the lymphatic glands; but suddenly laryngeal symptoms supervene, and death rapidly follows from suffocation; and the disease may be confounded with primary croup, if the pharynx has not been examined.

In the *nasal form of diphtheria* a sanious discharge from the nose attracts attention, after some febrile disturbance of a low type. The glands about the angle of the jaw begin to swell, the arches of the palate and tonsils become red and swollen, muco-purulent fluid bubbles in quantity from the narrowing isthmus of the fauces, and is apt to prevent the physician from seeing clearly the state of the pharyngeal mucous membrane. After a few days the disease may subside so completely as to leave its nature doubtful; but it may, on the other hand, spread to the larynx or the pharynx, when laryngeal or pharyngeal symptoms prevail, and the diagnosis is easy.

In *primary laryngeal diphtheria* the disease begins with painful deglutition, and is attended with redness and swelling of the mucous membrane of the pharynx, arches of the palate, uvula, and soft palate. Laryngeal symptoms rapidly supervene; and lymph may be seen on the arches of the palate, the exudation being more abundant at the base of the arch than above it, looking as if it spread from the larynx. Death threatens from *apnoea*.

In the *asthenic form of diphtheria* the patient dies from the constitutional effects of the general disease, which may begin with general and local symptoms of very moderate severity. The pulse, however, soon becomes rapid and feeble; the sense of weakness and of illness becomes extreme; the skin has a feverish pungency of heat to the touch; the complexion assumes a dirty-looking, pallid, and opaque aspect; and from an early period of the disease the tongue is brown, with *sordes* on the teeth. More or less lymph may be seen on the pharyngeal mucous membrane; and this lymph is of a granular, pulpy, or soft form. The patient may also swallow with perfect facility, and the throat symptoms may appear to be trivial in degree, even when the pharyngeal mucous membrane is covered with lymph. In some cases, however, the pain on swallowing is extreme. The exudative process may extend to the larynx, and this extension is indicated by a little huskiness and want of power in the voice, and imperfect laryngeal breathing. Death tends to supervene by asthenia about the tenth or twelfth day of the disease, preceded or not by delirium, which may commence at an early period of the disease.

There is no sharp line of distinction, however, between these several varieties of diphtheria.

The duration of cases of diphtheria varies from forty-eight hours to fourteen days; and when the disease is fatal within a week, it is so by extension of the exudative process to the larynx. Laryngeal symptoms rarely commence after the expiration of the first week of the disease; and in more than half the fatal cases of diphtheria death results directly from disease of the larynx. When death occurs from asthenia the fatal result usually takes place about the second week of the disease. In the cases that are not fatal the spe-

cific disease terminates between the eighth and fourteenth day of the illness (JENNER).

Prognosis.—However mild a case of diphtheria may appear to be, no case is unattended with danger. The great danger during the first week is from extension of the exudative process to the larynx; and the least laryngeal quality in the respiration heard at the bedside is suggestive of danger. Subsequently to the first week death is to be apprehended from exhaustion and loss of nervous energy. An extremely rapid and feeble pulse is of grave import; and a very infrequent pulse is of fatal significance. Vomiting is another unfavorable symptom, especially if it should recur many days in succession. Hemorrhages and albumen in the urine indicate blood change of great severity; and if the albumen is abundant, a fatal termination of the case may be expected. All the cases in which Dr. Jenner has known delirium to occur have ended fatally.

The danger in diphtheria seems to be in proportion to the youth of the patient. In the child, death is generally due to the extension of the disease to the larynx; after puberty it more often occurs from the general affection.

Sequelæ.—After the termination of the disease, symptoms of a very peculiar and characteristic kind are apt to supervene. The phenomena are referable to deranged innervation; and although their frequency and intensity are by no means invariably proportional to the severity of the primary disease, yet the more severe the case is, the more likely is nervous disorder to occur, and the more intense is it likely to prove. These consecutive phenomena do not appear at once. There is usually a brief period of convalescence between the disappearance of the primary and the appearance of the secondary phenomena of diphtheria. This period of temporary convalescence varies from a few days to a few weeks. The most alarming symptoms are referable to the heart. The frequency of its beats per minute begins to diminish, and a sense of languor supervenes, with tendency to vomiting. The heart's beats are found to be feeble, infrequent, and slow, and death supervenes from cessation of the heart's action (JENNER); or suddenly, from the deposition of fibrine within the heart, or in one of the great vessels (TANNER).

In other cases the paralysis is more widely spread, and the nervous symptoms more striking; although the nerve affections do not at once attain their maximum of intensity, but are progressive; and the progress of the paralysis, even in the same set of muscles, is seldom quite uniform. It is believed that the paralysis is due to a primary peripheral alteration of the nerves, which is propagated from the originally affected part to the spinal centre, much in the same way as in tetanus the irritation is transmitted from the wound (WEBER). If several sets of muscles are attacked, the faucial or pharyngeal are usually the set to suffer; and the impairment of function is very early betrayed by the condition of the voice, and by the act of swallowing, with loss of sensibility of the *velum pendulum palati* (TROUSSEAU). The sight is subsequently apt to become impaired; then the muscles of the tongue, the lips, and those of the

upper and lower extremity, become affected in the order named. In Dr. Greenhow's experience anæsthesia has coexisted with the paralytic affection of the fauces in all the cases. The mucous membrane over that region, naturally so sensitive, becomes altogether insensible and callous, even to repeated and forcible pricks with the nib of a pen; and in rare cases speech becomes so inarticulate as to be almost unintelligible. He has also observed that the paralysis and anæsthesia are sometimes more complete on that side of the fauces which was most severely affected in the early stage of the disease. When the pharyngeal muscles are paralyzed there is alarming difficulty in swallowing. The impairment of vision is probably due to paralysis of the ciliary muscle. It generally comes on suddenly, and is preceded for a day or two by dilatation and sluggish action, or actual paralysis of the iris. The tongue, lips, and cheeks may also become affected, both as regards motion and sensation. The parts then become numb or cold, or they experience a sense of formication or of scalding, and taste may be lost. The upper extremities are either first affected, or simultaneously with the lower. The power over the movements of the limbs becomes impaired, and anæsthesia is also more or less complete; or there may be tenderness with abnormal sensations, such as formication, and a perception of constriction or of tightness in the fleshy parts; or there may be convulsive movements of the limbs, resembling chorea. The earliest indications of these phenomena connected with the functions of the limbs are peripheral. Tingling is experienced at the tips of the fingers, accompanied with numbness, rendering the patient unable to pick up small objects. These phenomena then extend gradually to the wrist, and upwards to the elbows, and even to the shoulders; but the tingling and numbness are especially felt on the palmar surface of the hands. The limbs, when so affected, feel heavy, feeble, and cold. If the paralysis continues, the muscles concerned become flabby and emaciated, and strength becomes so much impaired that patients, who can move the affected limbs freely in bed, often walk with much difficulty, or are unable to stand unless supported. Increased sensibility of particular parts of the affected limbs accompanies the loss of power and general numbness, causing great distress, such as tenderness of the soles of the feet, and calves of the legs, or fleshy parts of the arms. There is also pain on pressure along the course of the principal nerves, such as the median nerve of the arm and the sciatic of the leg (*Edin. Med. Journal*, August, 1863). The duration of the paralysis varies greatly, but generally the cases eventually terminate favorably; the paralysis being only dangerous when it affects the respiratory muscles, and aggravates thereby an intercurrent pneumonia.

These phenomena of impaired nervous power generally betray themselves within three weeks from the date of convalescence; and the longest period at which Dr. Jenner has known death to occur after the first symptoms of diphtheria has been about two months.

Propagation of Diphtheria.—The disease seems to be infectious;

and family constitution (rather than any anti-hygienic conditions) favors its development and determines its progress (JENNER, GREENHOW, SANDERSON).

Treatment.—So long as there is heat of skin and firmness of pulse the physician ought to abstain from alcoholic stimulants, and rest contented by giving such saline medicines as exert a slight action on the skin and on the kidneys, or on both. *Acetate of ammonia* and *citrate of potash* are well suited for this purpose. The bowels should be opened freely by a dose of *calomel* and *jalap*; or by *calomel* and *colocynth* pill, followed in the inflammatory or *sthenic* forms of the disease by a saline aperient—*e. g.*, *sulphate of magnesia* in the *infusion of roses*.

The throat affection should be treated with warm fomentations externally, and by the inhalation of water vapor with acetic acid. A wine-glassful of vinegar to a pint of water is a good proportion (JENNER), and an inhaler should be used, as mentioned at page 317, under scarlet fever. Dr. Jenner recommends Squire's inhaler as the best. A lead gargle may be of service, composed of one fluid drachm of the solution of diacetate of lead in eight ounces of rose-water; but gargles must not be persisted in if pain is caused by their use. The temperature of the room in which the patient is confined to bed ought to be kept at 68° Fahr., and its atmosphere made moist by the steam from a kettle with a long spout constantly boiling on the fire. If the patient can be enveloped in a warm moist atmosphere, so much the better; and this may be done by making a tent with blankets over the bed, and, by the aid of a spirit-lamp, a tin kettle of boiling water may be maintained at the boiling-point, and its steam thus made to envelop the patient.

If feebleness of pulse supervene, if the redness of the throat assume a dusky hue, if the sense of general weakness become extreme, wine in large doses frequently repeated is required. Six or eight ounces of port or sherry during the day for an adult may be given from the first, with as good a diet as the stomach can digest. During the course of the disease, much larger quantities of wine and even brandy may be necessary; but the quantity of stimulants must be regulated by the habits and age of the patient. A child of three years of age may take with advantage one or two drachms of brandy every hour—*i. e.*, from three to five ounces of brandy during the twenty-four hours (JENNER). Under all circumstances efficient daily action of the bowels must be secured, and the urinary and intestinal secretions should be examined daily.

If blood or albumen appear in the urine, diuretics are contraindicated. Mustard poultices, warm linseed-meal poultices, or the warm wet sheet, as recommended by Dr. Huss in typhoid fever, and referred to at page 387, may be applied to the loins under these circumstances. *Tincture of the perchloride of iron* has been recommended by Dr. Hilslop, of Birmingham; and it may be advantageously combined with quinine in the following formula (TANNER):

R. Quinæ Sulphatis, gr. ij; Acidi Hydrochlorici diluti, ℥x; Tincturæ Ferri Perchloridi, ℥xv; Infusi Calumbæ, ʒj; *misce. Fiat haustus, omnibus sextis horis sumendus.*

Copaiva and cubebs are also highly spoken of by Frideau and by Trousseau.

With regard to topical applications, Dr. Jenner is of opinion that repeated applications to the throat of caustic solutions are injurious. He recommends one single but efficient application of a strong solution of nitrate of silver (℥j to 5j of water), as a remedy which may stay the spread of the exudative inflammation; but that, on the whole, *hydrochloric acid* and water in equal parts will more frequently attain the object. It is especially the surface round the exudation, as well as the exudation itself, that should be painted well over with the solution, the brush being passed over the surface two or three times in quick succession. The white discoloration which results must not be confounded with the spread of the diphtheritic exudation. The discoloration from the acid passes away in about thirty-six hours; and that from the nitrate of silver somewhat quicker. *Medicinal carbolic acid* (as prepared by Calvert, of Manchester), is valuable as a gargle, in the proportion of 1 of acid to 200 parts of water.

There is considerable difference of opinion regarding the usefulness of topical applications, and the best means of applying them. The tincture of the perchloride of iron is recommended by some to be gently painted over the fauces. The local application of lime-water by frequent gargling or gentle brushing, [or by atomized spray,] with the internal administration of nitrate and carbonate of soda, prove speedily curative in the milder cases, and alleviate the more severe. Ice kept dissolving in the mouth is often also a great comfort in cases of diphtheria, of croup, and of all sore throats, and its use should never be omitted where it can be had. Dr. Greenhow remonstrates against the application of the more severe topical remedies. The pellicle or false membrane ought never to be torn off. In the consecutive paralysis tonics and local galvanism are the most important remedies, and the bowels should be kept open by a pill, taken morning and evening, containing from a quarter to half a grain of the extract of *nux vomica*, with a like quantity of sulphate of iron, combined with two or three grains of compound rhubarb pill mass. These may be varied with the administration of pills containing $\frac{1}{2}$ th of a grain of *strychnia*, the *strychnia* being triturated with sugar of milk, and made into pills with a sufficient quantity of extract of gentian. Syrup of the phosphate of iron in fluid drachm doses may be given twice a day, and stimulants in the form of malt liquors, especially stout (if free of *cocculus indicus*), are beneficial if taken with or after meals, and the doses of iron may be taken at the same time.

Tracheotomy undoubtedly saves a small proportion of cases. It ought to be had recourse to if the exudative inflammation extends to the larynx and advances in severity. The degree and increase of the recession of the soft parts of the parietes of the chest during inspiration is the guide to its necessity. In the adult, laryngotomy is to be preferred to tracheotomy. Dr. Jenner is of opinion that the opening should be made even through the seat of disease, and not below it; for, in opening through a healthy part, a new centre of irritation and inflammation is established. The sole object contem-

plated by an opening in the windpipe is the prevention of death by suffocation. By so averting death, time is gained for the general disease to run its course (JENNER).

CROUP.

LATIN, *Angina trachealis*; FRENCH, *Croup*; GERMAN, *Croup*—Syn., *Häutige Bräune*; ITALIAN, *Laringitide membranacea*.

Definition.—A specific disease, accompanied by the exudation of an albuminous material upon the mucous membrane of the epiglottis, glottis, larynx, or trachea, and sometimes over all of these parts, indicated by accelerated, difficult, wheezing, or shrill respiration; short, dry, constant, barking cough; voice altered by hoarseness, with spasm of the interior laryngeal muscles, and pain and constriction above the sternum; frequently followed towards the close of the disease by expectoration of a membranous albuminous substance, or even of a cylindrical cast of some portion of the breathing-tube. The disease occurs in children, and may terminate fatally either in suffocation or exhaustion of the vital powers.

Pathology and History.—It has often excited much surprise that a disease so distinctly marked in its symptoms should not have been accurately described before the middle of the eighteenth century, when Dr. Francis Home published a treatise on the *suffocatio stridula* or *croup*, in 1765, as it was observed in Leith, Musselburgh, and the vicinity of Edinburgh. It has been described under the name of *cynanche trachealis*; and Dr. Farr has proposed for it the name of "*trachealia*" in scientific nosological nomenclature.

Before the time of Dr. Home, however, there is reason to believe that the disease was confounded with other affections of the throat and breast resulting simply from exposure to cold. It was certainly also described and distinguished by Martin Shisi, in 1749, at Cremona, and by Starr, of Liskeard, in Cornwall, in the same year (*Phil. Trans.*, 1750). Many physicians have described the disease since that time, and none with more minuteness than Dr. Cheyne, of Leith, who observed it for several years, and illustrated its pathology by careful dissections.

The most remarkable pathological phenomena of croup are to be observed in the exudative process which attends the inflammation in the windpipe, and the formation of a false membrane, almost peculiar to children, but sometimes seen in adults. When death takes place after an illness of four or five days, the windpipe is found to be lined with a white or gray substance. The membranes thus formed vary much in thickness and consistency. Some are so thin that the mucous membrane is readily seen through them, while others are many lines in thickness, exceeding even that of the mucous membrane itself, and consequently opaque. With respect to their consistency, some are so little coherent that they are almost diffuent, while others can be detached for a considerable extent without rupturing. The false membrane, though occasionally only partial, yet more commonly embraces the entire circumference of the larynx,

forming a complete hollow cylinder adapted to the walls of the larynx. The membrane is in most instances limited to the larynx, but in some cases it extends down the trachea to the bifurcation, while in a very few cases it reaches even to the minutest branches of the bronchi. M. Hussenot says, of 120 cases he examined in 1778, it did not extend beyond the larynx, while in 42 cases it invaded the trachea or bronchi. The membrane thus formed is in a few instances removed by the cough, but more generally it adheres with so great tenacity that it can only be detached by a thinner and more serous secretion taking place from the mucous membrane beneath it, which loosens and displaces it. The extent of the exudation, as indicated by the surface covered, is perhaps the most interesting and practically useful part of the pathology of the disease. The place first and most particularly affected is the upper part of the trachea, about an inch below the *glottis*. In that part patients complain of a dull pain. External swelling has been observed there; and the morbid membrane is found spreading from that place downwards. The back part of the trachea, where there are no cartilages, seems to be its first and principal seat (HOME). According to Guersent, false membrane is never entirely absent from the larynx. Sometimes it is confined to the glottis, and sometimes lines the whole interior of the larynx, including the ventricles; and not unfrequently it extends throughout the trachea, and, for a greater or less distance, into the bronchial tubes. Dr. Wood instances a case in which he saw the false membrane line the upper portion of the bronchia, the whole trachea and larynx, and the pharynx as low down as the œsophagus. More frequently, he says, the exudation is in the form of patches, or long narrow ribbons, and occasionally, in the earlier stages, it has a granular aspect, with the red mucous membrane appearing in the intervals of the imperfectly connected patches. According to Cheyne, in none of the cases recorded by him was membranous exudation observed in the laryngeal mucous membrane; and if the inflammation extended to this part, it was only slight, and its effects were seen in a little puriform fluid in the membrane of the cirroid or thyroid cartilages. Some state that it is essential to the constitution of croup that the larynx should be more or less involved in inflammation, or high vascular irritation, accompanied with spasms of the internal muscles of the larynx (WOOD). Others say that the inflammation in croup is truly tracheal, and even bronchial (CRAIGIE, COPLAND). In the more acutely inflammatory form it may extend to the larynx and epiglottis, in some cases; in others, to the first ramifications of the bronchi; and sometimes in both directions (COPLAND).

Dr. Copland, who has paid particular attention to the pathology of croup, states the following as general inferences from his observations: "(a.) That the mucous membrane itself is the seat of the inflammation of croup; and that its vessels exude the albuminous or characteristic discharge, which, from its plasticity and the effects of temperature and the continued passage of air over it, becomes concentered into a false membrane; (b.) That the occasional appearance of bloodvessels in it arises from the presence of red globules in

the fluid when first exuded from the inflamed vessels, as may be ascertained by the administration, upon the approach of the symptoms, of a powerful emetic, which will bring away this fluid before it has concreted into a membrane; these globules generally attracting each other, and appearing like bloodvessels, as the albuminous matter coagulates on the inflamed surface; (c.) That the membranous substance is detached in the advanced stages of the disease, by the secretion from the excited mucous follicles, of a more fluid and a less coagulable matter, which is poured out between it and the mucous coat; and, as this secretion of the mucous *cryptæ* becomes more and more copious, the albuminous membrane is the more fully separated, and ultimately excreted if the vital powers of the respiratory organ and of the system be sufficient to accomplish it; (d.) That subacute or slight inflammatory action may be inferred as having existed, in connection with an increased proportion of fibro-albuminous matter in the blood, whenever we find the croupal productions in the air-passages; but that these are not the only morbid conditions constituting the disease; (e.) That in conjunction with the foregoing—sometimes only with the former of these in a slight degree—there is always present, chiefly in the developed and advanced stages, much spasmodic action of the muscles of the larynx, and of the transverse fibres of the membranous part of the trachea, which, whilst it tends to loosen the attachment of the false membrane, diminishes, or momentarily shuts, the canal (of the larynx) through which the air presses into the lungs; (f.) That inflammatory action may exist in the trachea, and the exudation of albuminous matter may be going on for a considerable time before they are suspected—the accession of the spasmodic symptoms being often the first intimation of the disease; and these, with the effects of the pre-existing inflammation, give rise to the phenomena characterizing the sudden seizure; (g.) That the modifications of croup may be referred to the varying degree and activity of the inflammatory action, the quantity, the fluidity, or plasticity of the exuded matter, the severity of spasmodic action, and to the predominance of either of these over the others in particular cases, owing to the habit of body, temperament, and treatment of the patient, &c.; (h.) That the muco-purulent secretion, which often accompanies or follows the detachment and discharge of the concrete or membranous matters, is the product of the consecutively excited and slightly inflamed state of the mucous follicles, the secretion of which acts so beneficially in detaching the false membrane; (i.) That a fatal issue is not caused merely by the quantity of the croupal productions accumulated in the larynx and trachea, but by the spasm, and the necessary results of interrupted respiration and circulation through the lungs; (k.) That the partial detachment of fragments of membrane, particularly when they become entangled in the larynx, may excite severe, dangerous, or even fatal spasm of this part, according to its intensity relatively to the vital powers of the patient; and that this occurrence is most to be apprehended in the complicated states of the malady where the inflammatory action, with its characteristic exudation, spreads from the fauces and pharynx to the larynx and

trachea, the larynx being often chiefly affected in such cases, and from its irritability and conformation giving rise to a more spasmodic and dangerous form of the disease; (*l.*) That the danger attending the complications of croup is to be ascribed not only to this circumstance, but also to the depression of vital power, and the characteristic state of fever accompanying most of them, particularly in their advanced stages; (*m.*) That irritation from partially detached membranous exudations in the pharynx, or in the vicinity of the larynx or epiglottis, may produce croupal symptoms in weak, exhausted, or nervous children, without the larynx or trachea being themselves materially diseased; and that even the sympathetic irritation of teething may occasion the spasmodic form of croup, without much inflammatory irritation of the air-passages, particularly when the *prima via* is disordered, and the membranes about the base of the brain are in an excited state; (*n.*) That the predominance in particular cases of some one of the pathological states noticed above (*g.*) as constituting the disease, and giving rise to the various modifications it presents, from the most inflammatory to the most spasmodic, may be manifested in the same case, at different stages of the malady, particularly in its simple forms, and in the relapses which may subsequently take place; the inflammatory character predominating in the early stages, and either the mucous or the spasmodic, or an association of both, in the subsequent periods; (*o.*) That the relapses, which so frequently occur after intervals of various duration, and which sometimes amount to seven or eight, or are even still more numerous, may each present different states or forms of the disease from the others; the first attack being generally the most inflammatory and severe, and the relapses of a slighter and more spasmodic kind; but in some cases this order is not observed, the second or third, or some subsequent seizure, being more severe than the rest, or even fatal, either from the inflammation and extent of exudation, or from the intensity and persistence of the spasmodic symptoms,—most frequently from this latter circumstance. The above inferences, however minute or trite they may seem, should not be overlooked, as they furnish the safest and most successful indications of cure, and are the beacons by which we are to be guided in the treatment of the disease."

The name of *croup*, by which this disease has hitherto been known in this country, is of Scottish origin. Cullen's *cynanche trachealis*, and the more modern *tracheitis*, are objectionable terms, because they lead to false notions of the pathology of the disease. The "choak," "stuffing," "rising of the lights," and "hives," are all designations by which the disease has been described, and some of them are still names in vogue amongst the common people of the country.

The disease is almost peculiar to infancy and childhood; and there are two forms which can generally easily be distinguished from each other, but which are often confounded. One form is very manageable, the other very fatal. In the former variety the mucous membrane chiefly secretes mucus, pus, or muco-purulent fluid. In the more dangerous form an albuminous, fibrinous, or mucinous

exudation grows upon the inner surface of the air-passages, constituting the false membranes already described. The first form seems to be the one common in America, of which not more than *one* in *fifty* dies. The latter is the more common European form, of which the deaths used to be *four* out of *five*, and still are about a *half*. About one child in twelve deaths of children dies from this disease; and the ratio borne by croup to 1000 deaths from all causes, in 1854, was as 9.249.

Symptoms and Course.—The mildest form of croup differs from an ordinary catarrh only in the addition of spasmodic symptoms; but this form may run into the more severe form, so that it is not possible to determine, in the first instance, which form the disease may ultimately assume.

The catarrhal croup of Dr. Wood embraces the spasmodic as well as the catarrhal croup of Dr. Copland. Spasmodic action of the laryngeal muscles is, however, common to both, and is characteristic; but the inflammation and exudation is not in general more severe than that which attends a common catarrh.

The disease may be ushered in by sore throat, by catarrhal symptoms, or by a short dry cough; or it may occur *per se*, and without the general health being sensibly impaired. In either case the attack commonly takes place during the night, the sleep of the child, which was perhaps more or less agitated, being interrupted by fits of *hoarse* coughing. These become more frequent, the respiration more difficult, and marked by a peculiar wheezing, which has been described as like the sound of an inspiration forcibly made with a piece of muslin before the mouth, or like to the sound of air passing through a brazen tube. The little patient also feels a sense of restriction about the throat, as shown by carrying the hand often to it, and grasping the larynx. After the paroxysm has lasted some hours, there is an interval of ease, which perhaps lasts for some hours.

By the end of the second or third day, sometimes sooner, the tongue becomes white, the heat of the body increased, the pulse frequent, the face flushed, and the countenance distressed. From this point the disease now rapidly advances, the croupy sound attains its height, and Dr. Home describes it as "*vox instar cantus galli*;" others have compared it to the noise which a fowl makes when caught in the hand; while the child often puts its fingers into its mouth, as if to pull away something which obstructs the passage.

As the disease draws towards a close the paroxysms become more frequent, the cough more severe, the pulse more rapid, suffocation more imminent, and the extremities cold and livid. The final close of the disease is often by convulsions, sometimes almost tetanic; and Dr. Ferriar once was present when the struggle was so violent that after death the corpse, in a great measure, rested on the occiput and on the heels.

Often, however, the symptoms are much more moderate; although it not unfrequently happens that symptoms of the severer form come on, indicated by a huskiness of the voice, till no sound can be

heard above a whisper, by a muffled cough, and a wheezing noise which attends the inspirations. It is seldom that children expectorate; but in happier cases than the above, mucus, tinged perhaps with blood, is coughed up, and later, perchance, the false membrane is detached and thrown up, and the patient recovers.

The croup which has been described is of the most acute kind; but in many cases its course is much more chronic, the symptoms generally milder, and the intervals of ease longer and more complete, during which the breath is free, the child cheerful, and the appetite good. In the course of a few days, however, a violent paroxysm seizes the child, and destroys him with every appearance of one strangled.

According to Barth, on the stethoscope being applied to the larynx, we hear a sort of "*tremblement*," as if a movable membrane was agitated by the air; and he considers this phenomenon as an unerring evidence of the existence of a false membrane in the larynx. Laryngitis in the adult is marked by the same difficulty of breathing, the same lividity of countenance, the same constriction of the throat, the same paroxysmal attack, and by the same exemption from any severe constitutional affection. The voice, however, instead of being sharp and shrill, is generally deep and hoarse, although sometimes altogether lost—differences depending perhaps on the greater size of the glottis, and on the fact of the parts being the seat of ulceration rather than of the effusion of lymph. At length the patient is cut off in one of the paroxysms. The duration of this disease, when acute, is short. The celebrated Dr. Pitcairn died on the fourth day from the first attack, and Sir John Hay, Physician to the Forces, died within the same period. More commonly, perhaps, the disease passes into a chronic state, when the patient may survive many weeks, or even months. Several cases are on record of croup having terminated in twenty-four hours; more frequently, however, the child lives to the third or fourth day, and in chronic cases much longer. From one day to one or two weeks may be given as the variable periods of the duration of this disease.

Diagnosis.—It is generally between croup and the following diseases: namely, the different forms of sore throat, as in *scarlet fever* and *measles*, *diphtheria*, *bronchitis*, *chronic laryngeal and tracheal inflammation*, and *whooping-cough*; and the differential symptoms of each of these from croup must be studied by comparing the definitions, symptoms, and course of each of these diseases, as well as the epidemic constitution as regards *scarlet fever*, *measles*, *diphtheria*, and *whooping-cough*.

Modes of Propagation.—Croup is said to be more frequent in cold and moist climates than in those which are warmer. It is also much more severe in Europe than in America; and its existence and progress is considerably influenced by changes of season, weather, and temperature. It is prevalent in Switzerland and Savoy, in the eastern counties of England and Scotland, the northwest countries of Europe, and in the northern parts of America. While the annals of medicine are rich in descriptions of epidemic and endemic

croup, opinions are very much divided as to the nature of the epidemic influence, and whether or not the disease is contagious or infectious.

Age has, perhaps, the greatest influence in predisposing to the disease, and, while rare in adults, it is seldom seen in early infancy. It is most prevalent between the *first* and *seventh* years of life. According to the experience of Dr. Wood, the disease appears to run in families; and vigorous fleshy children, with rosy complexions, are frequently those who suffer most.

Prognosis.—"Is never better than doubtful." It is to be determined from the violence of the local symptoms, and the frequency of the paroxysms, rather than from the constitutional symptoms. Children, however, seized with croup are said to recover in a smaller proportion in this country than in America. Death tends to occur by *apnœa*.

Treatment.—*Every case of croup demands the most active, efficient, and energetic treatment.* When the croup in children commences in the larynx, its course is so rapid and so fatal that the measures for its suppression must be early. Bleeding, and especially local bleeding, should be employed, and in most cases to a considerable extent (an ounce of blood for every year of age); and two to twelve leeches, according to the age of the patient, should be applied over the larynx. After these have fallen off, the bleeding should be encouraged by the application of a linseed poultice to the throat. This first bleeding often gives great relief, and sometimes averts the disease; but if not, the leeches, after a few hours, may be repeated. As soon as some relief is obtained, a blister should be applied along the lateral aspect of the neck on each side, and not over the trachea; and after that is removed, the part should be dressed with strong mercurial ointment. In addition to bleeding and blistering, many practitioners prescribe *emetics*; first, because their emetic effects, and the large evacuations they produce, favor the resolution of the inflammation; and again, because the effort of vomiting may be the means of detaching and of expelling the false membrane, should it have formed. If relief does not ensue on the action of the emetic, Dr. Cheyne recommends *two, three, or four* grains of calomel, with *two or three* grains of James's powder, to be given at short intervals every two or three hours; and a dose of castor oil is to be given occasionally till the full effect of the calomel as a purgative is obtained. Green fecal stools, like chopped spinach, are characteristic of this result.

Bleeding, blistering, and mercury, although the rule of treatment in idiopathic infantine croup, are, for the most part, entirely inefficient in those cases in which the affection begins in the fauces, as in the case of many epidemics, and especially after scarlatina. In these cases the best treatment, if the false membrane be not already formed, is to relieve the throat by the application of a few leeches, as in scarlet fever, and then to support the little patient with a moderate quantity of wine diluted with water. If the false membrane has formed, perhaps an emetic affords the only chance of relieving the patient; and, indeed, so soon as croupy cough and

dyspnoea occur, an emetic of *ippecacuanha* with *tartar emetic* ought at once to be given in doses suited to age. Four to six grains of *ippecacuanha*, combined with a quarter or a third of a grain of *tartar emetic*, will be found sufficient for a child of two or three years of age. The action of the emetic may be aided, with benefit, by a warm bath of 98° to 100° Fahr. in temperature. If it becomes obvious that the exudation has assumed the form of a membrane, especially if indicated by a diphtheritic coating over the fauces, a solution of the nitrate of silver, varying in strength from forty to one hundred and twenty grains to the fluid ounce of distilled water, should be applied to as much of the fauces and larynx as can be reached. A sponge on the end of a piece of whalebone, as sold ready made by the instrument makers, should be loaded with the weaker solution, and squeezed against the *rima glottidis* two or three times a day. Bleeding has no effect in removing or modifying the false membrane; but the system must be brought as speedily as possible under the influence of mercury. Mercury appears a powerful resource in these cases; and, introduced either internally or by inunction, so as to affect the mouth, but without inducing salivation, uniformly gives relief as soon as the constitutional affection is established. However, the amelioration is too often transitory.

Expectorant medicines should be given with the mercurials, and be continued after them. *Ipecacuanha* and *seneka* have the most efficient influence over the mucous membrane. Five-grain doses of *iodide of potassium* every two hours, and of *chlorate of potassa* have been used with benefit; and the use of a vapor bath from 75° to 80° Fahr. is not to be neglected (Budd).

[The writer believes that bloodletting, either general or local, is generally harmful in the treatment of croup, as the disease is met with in the United States. The safest and best emetic is sulphate of copper, given in half- or one-grain doses every fifteen minutes, till vomiting happens. Afterwards give the following, in a little water, every second or third hour:

R. Potass. Chlorat., gr. v; Potass. Iodidi, gr. j; Tinct. Opii Camph., ℥v; Liq. Potass., ℥j-ss.

R. Brush the throat and upper part of larynx with a solution of one part of muriated tincture of iron and two parts of water. Beef tea, wine, quinine, and iron, are generally required.

Dr. Abeille speaks highly of inhalations of cinnabar. He throws into an uncovered vessel of boiling water, containing some mucilaginous flowers or roots, about 30 grains of cinnabar, every three or four hours. Every now and then he lets the child inhale the vapor. He recommends *ippecacuan* in vomiting doses, and gives nourishing food, and wine and water.]

The medical treatment of croup is so frequently unsuccessful that tracheotomy has often been had recourse to as the means of prolonging life, and, consequently, as affording an additional chance of the patient's recovery. Guersent has performed this operation repeatedly at the Hôpital des Enfants, but almost always without success. On the other hand, M. Trousseau states that he has saved one-third of his patients by its means; and of twenty cases Breton-

neau saved six. Perhaps the experience of the profession, generally, is equally discordant on this point at this moment; for those who operate early contend they save some portion of their patients, while those who wait till a case is advanced, and beyond medical treatment, before they resort to this measure, for the most part lose all their patients. The evidence, however, is daily accumulating which shows that tracheotomy ought to be resorted to much oftener, as a remedy for croup, than it has hitherto been, and that *at a much earlier period in the disease,—not as a last resource, when death from asphyxia appears imminent, and after treatment of the most depressing kind.* That this is the secret of success in France and in this country is shown by the experience of able physicians and good surgeons, of whom the names of M. Trousseau, the late Mr. Jones, of Jersey, Mr. Henry Smith and Dr. Fuller, of London, the late Dr. Cruickshank, of Dalmellington, in Scotland, and Mr. Spence, Professor of Surgery in the University of Edinburgh, Dr. George Buchanan, in Glasgow, and Professor Roser, of Tübingen, may be stated as authorities by experience. In country districts the performance of tracheotomy in a case of croup is almost imperatively called for in the majority of cases, *if some symptoms of amelioration do not follow the steady use of bleeding, emetics, the warm bath, and calomel purgation, pursued for twelve or sixteen hours.* I know that, in a wild country district of Scotland, where croup was very common and fatal, the late Dr. Cruickshank saved eight out of eleven cases during two years. A valuable paper by Mr. Smith, in *The Medical Times and Gazette*, 26th January, 1856; another by the late Mr. Jones, of Jersey, on the 8th November of that year; and, lastly, a paper by Dr. Conway Evans, in the *Edinburgh Medical Journal* for January and May, 1860, go to support the same conclusion,—namely, that an earlier introduction of air, by the operation of tracheotomy, for croup, would not only give a larger percentage of recoveries in this country, but would place the operation in the same favorable light in which it is now regarded in Paris and other parts of France. Tracheotomy in croup is undoubtedly gaining ground; and it cannot be denied that children perish in the first instance almost always from suffocation. Tracheotomy is therefore indicated in croup (as in diphtheria) as soon as there are urgent symptoms of obstruction of the glottis. When the respiration is so impeded that the demand for oxygen is only satisfied by difficult forced respirations, dreadful anguish is depicted on the reddened countenance covered with sweat; there is extreme restlessness; the patient tosses from side to side, gets out of bed one minute and into it the next, clutching spasmodically at those around him, as if seeking everywhere for help. *This is the proper period for the operation of tracheotomy in croup,—the time when success may be expected (ROSER).* Should the operation be longer delayed, symptoms of asphyxia appear, overloading of the blood with carbon ensues, the face suddenly becomes blue, with fixed and staring eyes, convulsive exertions are made, and anxious struggles for breath follow the stage of suffocative agony. In some cases the symptoms of asphyxia come on more slowly and are apt to make considerable progress

before the danger is fully appreciated. This insidious form of asphyxia is denoted by symptoms of great weariness and weakness, restlessness, oppression, anxious startings out of short slumbers, loss of consciousness and of feeling, approaching stupor, the face pale, and tending to become œdematous. If tracheotomy is delayed till these symptoms become expressed, it may still enable the child to breathe more freely, and thereby may promote the chances of recovery; but the child has usually no longer power to resist the advancing bronchitis. The causes of death after the operation are mainly pneumonia, bronchitis, or the severity of the constitutional febrile state (*Syden. Society Year-Book*, 1863, p. 278). Abscesses in the anterior mediastinum, pleuro-pneumonia, and pericarditis have been found after death. The fatal result, therefore, seems in some cases to depend on a cause acting generally on the system, and which ultimately destroys the patient. This cause gives a difference of type to the disease in this country from what it has in France; but as this difference appears, in the first instance, to be aggravated by the obstruction to the passage of air, there is thus a still more powerful reason why the operation of tracheotomy ought to be resorted to early. Age also seems to influence success to a considerable extent. Under *two* years of age few cases recover; but between the ages of *six* and *twelve* nearly one-half are saved (Coxway Evans).

[The following are the statistics of tracheotomy for croup, performed at the Hôpital St. Eugénie (Paris), for seven years and a half—from January, 1860, to July, 1867.]

Years.	No. of Operations.	Cures.	Proportion.
1860	55	8	12 per cent.
1861	76	13	17 "
1862	111	22	19 "
1863	112	34	30 "
1864	121	15	12 "
1865	147	46	31 "
1866	129	45	35 "
1867 (1st 6 mos.)	62	25	40 "
	813	208	

In seven years and six months, there were 971 cases of croup treated in the hospital, and in 813 of these tracheotomy was performed, with 208 recoveries. 158 cases were treated by medical means alone, and of these 94 recovered (*Gaz. Médicale*, No. 39, 1867).]

DYSENTERY.

LATIN, *Dysenteria*; FRENCH, *Dysenterie*; GERMAN, *Ruhr*—Syn., *Dysenterie*; ITALIAN, *Dissenteria*.

Definition.—A specific febrile disease, accompanied by tormina, followed by straining, or scanty mucous or bloody stools, which contain little or no fecal matter. The minute lenticular and tubular glands of

the mucous membrane of the large intestines, with the intertubular connective tissue, are the chief seats of the local lesion, which sometimes extends into the small intestine beyond the ileo-colic valve; as in cases in which scorbutus is a predisposing cause.

Historical Notice, Pathology, and Morbid Anatomy.—Dysentery is a disease which varies considerably in different countries and localities; and sometimes in apparent accordance with the exciting cause. Sporadic cases, which now and then occur in our large towns, are not generally so violent, and are less fatal than the epidemic cases, and those which occur in tropical climates. The effects on the constitution are no less varied and severe.

Dysentery has at all times proved one of the most severe scourges of our fleets on foreign stations, of our armies in the field, and during campaigns, even in temperate regions. It is sometimes so prevalent that it exceeds the number of sick from all other diseases put together. It has followed the tracks of all the great armies which have traversed Europe during the Continental wars of the past 200 years. It helped to destroy the British army in Holland in 1748. It decimated the French, Prussian, and Austrian armies in 1792. It was a chief cause of death in the ill-fated Walcheren expedition in 1809. It cut down the garrison of Mantua in 1811 and 1812. Sir James McGrigor records how fatal the disease was in the Peninsular campaigns; and we know how disastrous it was to our troops during the first winter they passed in the Crimea, in 1854. In the words of Sir Ranald Martin, "It is the disease of the famished garrisons of besieged towns, of barren encampments, and of fleets navigating tropical seas, where fruits and vegetables cannot be procured. During the Peninsular war, the first Burmese war, and the late war with Russia, *dysentery* was one of the most prevalent and fatal diseases which reduced the strength of the armies."

That it is a dangerous and frequent disease throughout our inter-tropical possessions, the tabular statements on the next page, furnished by Sir Alexander Tulloch to Sir Ranald Martin, and by Dr. Joseph Ewart, of Calcutta, sufficiently testify.

In England, generally, however, *dysentery*, as a cause of death, has been decreasing since 1852, although about 200 years ago it was one of the most prevalent and fatal diseases of London. Yet still, although the disease is less violent and less fatal (for as a cause of death it has remarkably diminished during the past ten or twelve years), and although the unfavorable hygienic conditions which were wont to bring about dysentery no longer exist, the active endemic conditions which favor, promote, or are congenial to its development, are only dormant, and not eradicated. The disease, therefore, is still sometimes brought about just as in the days of Sydenham or Willis. In no respect, however, do we find that the dysentery of this time differs essentially from the description given by Sydenham more than a hundred and thirty years ago. When we look, therefore, to the history of the disease, and to the nature of its lesions—to its reappearance from time to time among us, with the same identical characters—there are strong grounds for

PREVALENCE AND MORTALITY OF DYSENTERY IN VARIOUS COUNTRIES, BY THE
LATE SIR ALEXANDER TULLOCH, K.C.B.

STATIONS.	Period of Observation.	Aggregate Strength.	DYSENTERY.		
			Attacked.	Died.	Proportion of Deaths to Admissions.
Windward and Leeward command, }	20 years.	86,661	17,843	1,367	7.7 per cent.
Jamaica,	20 "	51,567	4,909	184	3.7 "
Gibraltar,	19 "	60,269	2,653	64	2.4 "
Malta,	20 "	40,826	1,401	94	6.6 "
Ionian Islands,	20 "	70,293	3,768	184	4.8 "
Bermudas,	20 "	11,721	1,751	36	2.0 "
Nova Scotia and New Brunswick, }	20 "	46,442	244	18	7.4 "
Canada,	20 "	64,280	735	36	4.8 "
Western Africa,	18 "	1,843	370	55	14.2 "
Cape of Good Hope,	19 "	227,111	1,425	44	3.0 "
St. Helena,	9 "	8,973	751	69	9.0 "
Mauritius,	19 "	30,515	5,420	285	5.2 "
Ceylon,	20 "	42,978	9,069	993	11.1 "
Tenasserim Provinces,	10 "	6,818	1,460	137	9.3 "
Madras,	5 "	31,627	6,639	559	8.3 "
Bengal,	5 "	38,136	5,152	411	8.0 "
Bombay,	5 "	17,612	1,879	151	8.0 "

AVERAGE RATES OF SICKNESS AND MORTALITY FROM DYSENTERY AND DIARRHŒA AMONG EUROPEAN TROOPS IN INDIA. (Compiled from Data contained in Tables XXVI and XXVII of *Vital Statistics of European and Native Armies in India*, by Dr. Joseph Ewart, of Calcutta Medical College.)

PRESIDENCY.	Periods.	Strength.	Admissions.	Deaths.	Percentage of Admissions to Strength.	Percentage of Deaths to Strength.	Percentage of Deaths to Admission.
I.—FROM DYSENTERY ALONE.							
Bengal,	1812 to 1853-4	543,768	100,542	8,873	18.48	1.64	8.82
Bombay,	1803-4 to 1853-4	306,978	51,010	4,705	16.61	1.53	9.22
Madras,	1829 to 1851-2*	213,587	30,593	2,304	14.32	1.07	7.53
II.—FROM DIARRHŒA.							
Bengal,	1812 to 1853-4	543,768	64,823	2,141	11.92	.39	3.30
Bombay,	1803-4 to 1853-4	306,978	32,290	551	10.51	.17	1.77
Madras,	1829 to 1851-2*	213,587	19,458	353	9.11	.16	1.81
III.—FROM DYSENTERY AND DIARRHŒA—AS A CLASS.							
Bengal,	1812 to 1853-4	543,768	165,365	11,013	30.41	2.02	6.65
Bombay,	1803-4 to 1853-4	306,978	83,300	5,256	27.13	1.71	6.30
Madras,	1829 to 1851-2*	213,587	50,051	2,657	23.43	1.24	5.30

* Exclusive of 1839, 1840, and 1841.

believing that there is something specific in the nature of the poison which produces dysentery, just as specific as that of *small-pox*, *typhus fever*, *typhoid fever*, *yellow fever*, *scarlatina*, *ague*, or *diphtheria*. But besides the specific identity of the disease, as it now exists, with the disease of former times, there is another point of view from which the history of the pathology of dysentery is especially instructive. It is this: Like all diseases which have been at the same time epidemic and severe, it has been the subject of discussions as frequent and as varied as its ravages have been severe; and one single description of the disease will not do for a record of the characteristics of all epidemics. Most minute descriptions of the state of the intestines in dysentery have been given by many writers; but, as Dr. Copland justly observes, from his extensive experience, "Dysentery is neither so simple in its nature, nor so unvarying in its seat and form, as most recent writers in this country have stated;" and "that writer will but imperfectly perform his duty who, in giving a history of a most prevalent and dangerous malady, confines himself to the particular form it has assumed during a few seasons, within the single locality, or the small circle of which he is the centre, and argues that it is always as he has observed it."

Dysentery is, moreover, a most formidable disease, on account of its oftentimes insidious nature, from its tendency to recur, and from the after-influences it exerts on particular organs and on the system at large. For these reasons almost all writers on the diseases prevalent in tropical climates place dysentery at the top of the list of severe affections, and refer to it as the cause and origin of many of those chronic and intractable abdominal diseases which so often afflict Europeans resident in tropical climates; and which entail most varied forms of impaired health when they return to European climates.

The morbid anatomy of dysentery has not been described with uniform distinctness, and the anatomical descriptions have, in general, been extremely vague. Medical science has not yet finally settled many points in the pathology of the disease; consequently, the doctrines as to treatment are somewhat uncertain, while the means of prevention are not less imperfectly defined. It has been usual to describe cases of *dysentery* as being either *acute* or *chronic*; but there are also cases belonging to a third class, which may be termed *complex*.

Acute Cases of Dysentery.—In this form the specific lesion in the form of inflammatory action does not confine itself to the tissues of the mucous membrane only. The serous covering of the intestines, or even such solid viscera as the liver, spleen, kidneys, are involved in a disease-process. Ulceration or sloughing of large portions of mucous membrane and exudation go on together, and there may be very little corresponding fever at all commensurate with the severity of the lesions, so that while the disease is acute, it is at the same time, in many instances, of a masked and almost latent nature. Death frequently takes place within the first ten or twelve days in such cases; but the disease may terminate gradually and spontaneously, or as the result of appropriate treatment, by the end of the third or fourth week. On the other hand, the disease may

not end so favorably and early; but, evincing a marked and obvious resistance to treatment, may advance unchecked; the morbid changes being slow in progress, often extending over several months, and then the case passes into

Chronic Dysentery.—One of the most hopeless and intractable forms of disease which the physician has to treat. Under the influence of the slow morbid changes about to be noticed, the wasting of the tissues of the patient progresses steadily, till a human form, literally reduced to the state of a living skeleton, whose bones are held together by skin and ligament, is all that remains. The skin acquires a dry, bran-like, furfuraceous aspect, and the epithelium desquamates in scales and powdery particles. During the progress of such chronic cases various intercurrent morbid states become developed, not necessarily connected with the primary affection, but forming secondary lesions to the disease, and constituting the third form in which *dysentery* must be studied, namely,—

Complex Cases of Dysentery.—There are various secondary lesions which render cases of dysentery complex, and which are regarded by some as directly connected with the primary affection. There are also secondary lesions connected with antecedent forms of disease, which sustain a renewed impulse to their development by the dysenteric state. These secondary lesions may be shortly stated to consist—(1.) In lesions of the small intestines, and of various solid viscera more or less connected with the dysenteric state; and (2.) In lesions which may be referred to the coexistence of certain morbid states of the patient with the dysenteric condition, such, for instance, as the *typhous*, *scorbutic*, and the *tuberculous* state.

Anatomy of the Morbid Tissues in Acute Dysentery.—The accounts of the morbid anatomy of dysentery are especially confusing; and while the disease has been mainly recognized during life, and defined as a febrile disease accompanied by tormina, and followed by straining and scanty mucous or bloody stools containing little or no fecal matter, yet the local lesions associated with these conditions have not been clearly defined nor uniformly described.

For example, Chomel and his school considered that the local lesion in dysentery consisted in congestion simply, and tumefaction of the mucous membrane, especially in patches of some extent, so as to form dark-red or purple prominences, from the surface of which the epithelium becomes detached by desquamation. Cruveilhier believed that dysentery was an erythematous inflammation of the large intestine, quickly followed by sphacelus; and he emphatically insists on the point that the follicles and solitary glands have no share in the disease. "It is not," he says, "a follicular inflammation." Rokitsansky includes these two forms of lesion as essential in dysentery.

The disease, as described by each of these observers, is regarded as a process of rapid, and at first of superficial, inflammation, leading inevitably and speedily to mortification, and unattended by any special disease of the solitary glands. Rokitsansky states that, even in the slightest variety of dysentery, the mucous membrane is swollen and red, and may be removed in the form of a pulp from beneath the furfuraceous and vesicular epithelium. In after stages,

and in the severer forms, the mucous membrane becomes gelatinous, and is easily separable, or it passes into a state of sphacelus, black, friable, and offensive. All these observers regard ulceration as having no essential part in the disease-process which constitutes dysentery, and as being of very rare occurrence.

Some of the writers who have described the tropical forms of the disease have been still less distinct as to the details of its morbid anatomy. For example, Twining seems to have followed Chomel in considering the lesion to be a simple inflammation of the mucous coat; and Annesley is in a great measure unintelligible as to the points of morbid anatomy which he describes. It was not till Dr. Parkes published his minute and admirable description of the morbid anatomy of dysentery, as he saw it in India, that we had anything definite on the subject as regards the tropical forms of the disease. He not only showed the very early implication of the glandular apparatus of the great intestine in dysenteric inflammation, but he established the fact, so far as his cases went, that, while ulceration occurs with great rapidity, a case never presents true dysenteric symptoms without ulceration being present. At Moulmein, in India, he investigated, in 1843-44, cases of dysentery in Europeans to the number of fifty, and in Asiatics to the number of twenty. He concluded from these observations that: (1.) Certain alterations in the glands of the mucous membrane of the large intestine, and sometimes of the ileum, constitute the earliest lesion in dysentery. (2.) That in all cases, when not too far advanced, the mucous membrane presented the appearance of numerous whitish round elevations, of a size varying from a millet-seed to a size so minute that a lens only can show the lesion. These elevations were hard, and being pierced, gave forth a white excretion. Many of these had a black speck in the centre, and were surrounded by a vascular circle. (3.) He noticed that exudation sometimes occurred in points beneath the mucous surface; that such points of exudation had a white appearance, with contents similar to those of the solitary glands. The mucous membrane over these points could be easily rubbed off, leaving an ulcer (Parkes *On the Dysentery and Hepatitis of India*).

The observations of Dr. Parkes were thus opposed to the views just stated, and led to extended investigation, by which such contradictions may be reconciled; and it was determined that differences of climate do not cause any essential difference in the structural changes which accompany dysentery. The observations of Drs. Craigie and Abercrombie in Scotland, in 1837, prove this; and also those of the late Dr. Baly, in 1847, as regards England. Drs. Cheyne, Graves, and Mayne have demonstrated the same fact as to Ireland. By the records of epidemic dysentery at Prague and elsewhere, as described by Dr. Finger and others, the observation holds true as regards the dysentery of Europe generally; and by comparing these records with the well-recorded cases of those who have seen the disease in the tropics, both in civil and in military life, it will be seen that the true dysentery of tropical and temperate climates does not differ as to its anatomical signs in any essential particular.

The descriptions of the disease in our own country, as given by

Cheyne, Craigie, Abercrombie, and Baly, all agree in recording the inflamed condition of the mucous membrane of the colon, with its small round ulcers, pulpy softening, or sphacelus of some portions, and ulcers of various forms left by the separation of the sloughs, and enlarged firm tubercles, which no doubt were the inflamed solitary glands. Again, Sir John Pringle, M. Broussais, and other historians of dysentery, found the same lesions in the dysentery of the camps in the continental campaigns of Europe; and Broussais expressly states his belief "that the ulcers of the large intestine had their origin in the solitary glands." Thus the "tubercles," the "pustules," and the "small-pox-like elevations" of the mucous membrane have been most minutely described by Hewson, Pringle, and Davis; and the last of these observers describes in graphic language the fatal dysentery of the Walcheren expedition, and shows that its anatomical characters are similar in all respects to the forms of dysentery which have been described in this country.

We have therefore only to compare all these records with the histories of tropical dysentery, as given by Zimmerman, Annesley, Pringle, Copland, Dr. Hunter, Chisholm, Ballingall, Parkes, Morehead, Sir Ranald Martin, Tait, Macpherson, and others, to know,— "whether any peculiar character of the anatomical changes in the large intestine essentially distinguishes the dysentery of intertropical countries from the dysentery of this and of other temperate regions." Dr. Abercrombie admitted identity in the nature of the dysentery; but, that extent of the intestine affected varies considerably. Dr. Craigie showed that the lesions in dysentery occur in two forms; one continues over the surface, the other limited to the *muciparous follicles*, which become enlarged, indurated, and ulcerated; while, on the other hand, Dr. Baly has shown that all the well-marked varieties of structural change in the large intestine occurring in tropical dysentery are likewise found in fatal cases of the disease occurring in our own country.

Seeing, then, that the descriptions of the morbid anatomy have been so much at variance with each other, several questions suggest themselves, namely,—(1.) Whether distinct epidemics are characterized by distinct local lesions? (2.) Whether two or more distinct diseases have not been confounded under the one name of dysentery? Or, (3.) Whether the various local lesions described by different writers are only so many varieties, forms, or types of the same disease-process—a process modified in particular cases by constitutional peculiarities, or by other circumstances. This latter view is the one most consistent with observation; and it is in accordance with what we know of the history of many of the *miasmatic* diseases, such, for example, as *true yellow fever*, *remittent fever*, *diphtheria*, and the like. There is some evidence, also, to show that there is a lesion of the colon not belonging to true dysentery—a *colonitis*, in which the connective tissue of the gut beneath the mucous membrane is implicated in the first instance, rather than the glandular tubes and vesicles. The result is a diffuse gangrenous inflammation of the mucous membrane, the resulting ulcers not differing from the ulcers originating in the glands by any characters at present recognizable (COPLAND, PARKES).

In this country it is believed that the lesion in dysentery is confined, for the most part, to the colon and rectum; but that in tropical dysentery the whole course of the colon, and sometimes a considerable portion of the small intestines, are implicated. But, except when the case is associated with *scorbutus*, the small intestines are not involved. Lesions so extensive, while they are common in India, are rare in this country; yet they do occur, and are not uncommon in the south of Europe, in Turkey, and the coasts of the Mediterranean. Therefore, as regards the extent of the lesion, there is no constant or distinctive characteristic between tropical dysentery and the dysentery of more temperate climates.

In both regions the anatomical changes comprehend redness of the mucous membrane, preceding further changes; loss of the substance of the mucous glands by pulpy softening of tissue, sloughing, or ulceration; the detachment of diphtheritic casts of the intestine, or sloughs of tissue.

In describing the morbid anatomy of dysentery the reader is referred to the nomenclature of the gland structures, given in a footnote at page 364 of the present volume. The structure of the colon in the healthy state differs in many important particulars from that of the small intestine. It is remarkable for the absence of folds and villi, and for the presence of more or less dilated sacculi, which give form and shape to the excrement. The minute tubular glands are thicker in proportion to their length, compared with those of the small intestine; and the intertubular connective tissue is considerable,—a structure which takes an important share in the lesions of dysentery. These tubular glands are lined by columnar, cylindrical, and transition forms of epithelium; and the solitary lenticular glands are sometimes closed vesicles (ALLEN THOMSON, PARKES, BALY), and sometimes open follicles. When closed they are not visible; but if distended, they may be seen with a lens; and when open, a dark depressed point marks the separation in the tubular gland structure which leads to the open follicle. The tubular glands radiate round this spot, which corresponds to a depression indicating the empty vesicle below. These solitary vesicles have thick walls, and are said to be more abundant in the cæcum and rectum than in any other part of the great intestine. This statement leads to the question which has been mooted in relation to these solitary gland lesions, namely,—“Are these lesions of the so-called solitary glands really due to the enlargement of previously existing solitary glands or their germs? or, Are they new formations altogether?”

A similar question is at issue regarding the granulations on the eyelids and conjunctivæ, associated with purulent ophthalmia (STROMAYER, FRANK, MARSTON). In this disease we have *new formations* of vesicular-like granulations, as well as enlarged follicles; but these are more numerous than the glands have ever been seen to exist in the healthy state. Observations somewhat similar have been made regarding the vesicular glands of the stomach (HANDFIELD JONES). It may be, therefore, that not a few of the “tubercle nodules,” the “pustules,” the “small-pox-like elevations,” and what we call solitary or lenticular glands, are in reality new formations altogether,

resulting from increased cell-growth, within the meshes of the connective tissue which binds the mucous gland-tubes together, and connects them with the submucous muscular layer. In this respect their formation would be analogous to that described by Virchow in connection with tubercle, and not to be distinguished histologically from a newly formed gray tubercle nodule. So independent have these lesions been believed to be by some, that one observer of Indian dysentery (MURRAY) described a "pustular form of dysentery," which he considered to be in all respects analogous to small-pox on the skin, beginning with the formation of an independent papule and the development of a subsequent pustule, as in that disease of the skin. This view of the subject is of some importance in pathology, as it is related to the specific nature of dysentery and the poison cast off from the mucous membrane, by which it is believed that the disease is propagated like typhoid fever and cholera (DR. WILLIAM BUDD).

Seeing, therefore, that the anatomical signs of dysentery are so constant over all the world, it may be asked, How have modern writers given such contradictory accounts of the morbid anatomy of the disease? The best writers have differed on points of observation simply: (1.) Some deny the necessary occurrence of ulceration. (2.) Some deny any special participation of the lenticular glands. (3.) Some believe that new formations arise, which are similar in appearance to those small glands. (4.) Some, on the other hand, believe the disease, at its commencement, to be always seated in these small glands. (5.) Some regard dysentery as essentially an erythematous inflammation, terminating in gangrene; (6.) Others believe that such gangrene is a very rare variety of the disease.

To explain such discrepancy, it may be said that: (1.) Observations have been too limited, and not exact enough, to give an accurate and comprehensive view of the morbid anatomy of the disease; that dysentery, although a simple and uniform disease, so far as its anatomical signs are concerned, yet it is liable to constant changes of type, from its remarkable proclivity to complicate prevailing fevers (specific or endemic), as well as other diseases (e. g., *typhoid fever, malarious fevers, fever from the poison of animal effluvia, scurvy, syphilis, phthisis, measles, variola*, and the like). (2.) The healthy or normal anatomy and histology of the mucous membranes are only yet beginning to be understood and studied minutely at our Schools of Medicine. (3.) The examination of the colon, upon which descriptions have been based, has often been incompletely done, as Annesley very correctly pointed out. (4.) The inherent difficulties of the subject itself, such as the impossibility of seeing the state of the diseased membrane till after death. From all the observations that have been made, there can be no doubt that the anatomical signs of true dysentery are primarily derived from inflammation of the solitary lenticular follicles of the large intestines, tending in the first instance to infarction (*i. e.*, intumescence and congestion), and subsequently to ulceration and destruction of the gland tissue. The disease, extending by a similar process, ultimately involves the tubular glands of the general mucous membrane, which tend to

soften and to be cast off as an exuvium or slough, exposing the submucous connective tissue or the muscular coat of the intestine. It is the mucous membrane of the great intestine, and especially of the rectum and lower portion of the colon, which is the seat of these characteristic lesions in dysentery. The exudative process is generally diffuse, involving the whole of the tissues of the mucous membrane. In some cases, however, it is seated in the solitary glands, in the first instance, and neighboring mucous tubular glands.

In the scorbutic form of dysentery, or in dysentery occurring in persons whose nervous or vital powers are feeble, or below *par*, as in the aged, infirm, or in the paralysis of the insane (conditions in some respects similar in their influence to *scorbutus*), a diphtheritic exudation covers to a considerable thickness not only the mucous surface of the colon, but (as an almost constant and pathognomonic morbid sign) the same lesion is seen covering the mucous surface of the small intestine, extending upwards from the ileo-colic valve; and, as Dr. Parkes has noticed, this exudation grows or is laid down especially in the course of the bloodvessels ramifying from the mesenteric attachment transversely across the surface of the gut, and occupying especially the prominences of transverse rugæ. This form of dysentery prevailed to a great extent among the soldiers who died at Scutari during the period of the Russian war when scorbutus prevailed. Some of the cases recorded by Dr. Davis, in his admirable description of the dysentery so fatal to the British troops in the famous Walcheren expedition, were also of this nature. Dr. Finger, of Prague, and Dr. Maine, of Dublin, and Dr. Baly, have recorded similar cases; but one of the best accounts of this form of dysentery is that which Dr. Clouston records as having prevailed in the Cumberland and Westmoreland Asylum, and which he believed to have been caused by the effluvia from a field irrigated with sewage. The regular diet of the Asylum inmates consisted of 24 ounces of animal food, 14 pints of milk, 16 ounces of suet dumpling, 7 pints of oatmeal porridge, 78 ounces of bread, and 7 pints of tea per week (*Med. Times*, June 3, 1865). No fresh vegetable diet seems to have been provided for; and diarrhœa does not seem to have been uncommon.

In typical cases of the outbreak described by Dr. Clouston—

“All the abdominal organs would be found healthy until the small intestine was examined. This, too, would be normal up to within five or six feet of the cœcum. The mucous membrane would then begin to appear reddened in small spots or rings round the gut. Six inches farther down the redness would be universal, and the membrane would begin to be thickened and corrugated into folds, like small *valvulæ conniventes*. A few inches farther down, a yellowish, dirty-looking deposit would be seen over the mucous membrane in rings, very thin where it began, but gradually becoming thicker and more continuous till near the cœcum it would be one-eighth of an inch in thickness. The swelling of the mucous membrane would also increase downwards, and the folds running across the gut would become more prominent. These, with their coating of deposit, made the inside of the bowel look like a series of thick transverse ridges,

covering its entire surface. This deposit, when examined, would be found to be soft on the surface, but getting more firm towards the mucous membrane, with which it incorporated itself, so that it could not be scraped off without leaving the fibrous covering of the muscular coat exposed as a highly vascular, raw-looking surface. This deposit, though on the surface of a soft lymph-like substance, yet towards the mucous membrane it assumed quite the consistence and appearance of a soft fibrous membrane. The cæcum, when examined in such a case, would be found in the same state as the lower part of the small intestine, with two or three ragged ulcerations the size of beans. In the ascending colon the ulcerations became deeper and larger; still the lymph deposit on the surface of the mucous membrane became thicker and more feculent in color. Towards the transverse colon the inside of the gut was one mass of large irregular ulcers, with patches of the deposit between them. The color of the surface was almost black, and this continued down to the very lower part of the rectum. The mesenteric glands opposite the affected parts of the small and large intestine were enlarged and dark-colored, and on section were soft and pulpy in consistency. Such were the general appearances in a case that had lasted for about a month" (*Med. Times and Gazette*, June 10, 1865, p. 598).

The morbid anatomical states which I have been able to distinguish throughout numerous dissections of cases of dysentery may be stated as follows:

1. Exudation obvious on the surface of the mucous membrane of the rectum and colon.
2. Exudation not obvious to the unaided eye, but which was seen, in all the cases examined by the microscope, to fill the mucous tubular follicles of the large intestine.
3. Exudation obvious to the eye, and demonstrable by microscopic examination as being developed in the solitary vesicular or lenticular glands of the large intestine.
4. Changes in the exuded material, which tends first towards its organization, and subsequently to its destruction and removal by ulceration.
5. Softening and ulcerative changes in the tissues of the mucous membrane itself, and in the glands.
6. Similar dysenteric lesions extending into the *small intestines*, as in the scorbutic states of the system.

The extent of the exudative process varies much. In some cases a considerable portion of the *colon* and *rectum* only is affected; in other instances not only is the whole of the great gut the seat of some form of the exudative process, but the lower portion of the small intestine also. The most commonly affected portions, however, are the *rectum*, the *sigmoid flexure*, and the *descending colon*. When the *caput cæcum* of the colon is involved, the *vermiform appendix* participates in the process. Creamy-like exudations have been seen to fill its tubular glands, which in some cases were opened up by ulceration.

In the least severe cases an opportunity does not often occur to see the changes in an early stage; but when life is cut short by some other malady, changes of the following nature may be seen:

The exuded mucinous material, in its more recent state, forms a layer, which varies from a thin but opaque membrane to three or four lines in thickness, of homogeneous substance, tolerably consistent, and capable of being detached and raised in flakes from the subjacent mucous surface. During the earlier stages of the disease the surface of the mucous membrane appears unchanged below, except, perhaps, by the existence of a little increase of vascularity. The color of the exuded matter may be uniform, or red, white, or pink in patches, and discolored in some instances by intestinal gases, the biliary secretion, or by the admixture of blood, and the changes consequent thereon. The most common appearances in severe cases is that of a dark olive-green, passing into a bluish-black. The surface of the exudation may be uniform, or the whole aspect may be mammillated, with here and there a *mammillation* projecting greatly above the others in a fungating mass, surrounded by dark fissures in the exudation. These fungating masses are soft towards their centres, with numerous red vascular points here and there on the surface. A section through the mass shows the base thickened and firm.

The dysenteric process, as seen after death, is generally found to have advanced farther in one part of the intestine than in another; usually, it may be stated to have been farther advanced in the *rectum* than in the *descending colon*, and farther in that part than towards the head of the large intestine. In well-marked and extreme cases the entire mucous surface, from the *caput cæcum* to the *rectum*, may be seen to present all the possible stages of the dysenteric process. Three stages can in general be distinguished, namely,—(1.) Ulceration of the exudation and mucous membrane more or less advanced towards the *rectal end* of the great intestine; (2.) Exudation in various forms towards the *middle* of the *colon* upwards from the *rectum*; (3.) The exudative process visible microscopically in the tubular glands, and sometimes also obvious to unaided vision in the solitary vesicular glands of the great intestine towards the *caput cæcum*.

One of the best descriptions of the morbid anatomy of dysentery in the English language has been given by the late Dr. Baly in his *Gulstonian Lectures* for 1847. He describes *three* different forms of lesions as seen by him amongst convicts at the Millbank Prison; and these three forms he believed to correspond with three degrees of severity of symptoms during life,—(1.) He recognized a swollen condition of the solitary glands, forming round prominences on the surface of the mucous membrane, of various sizes. In color these were pale, or red round the base, and dotted at the summit with a vascular spot. These appearances would occur about the eighteenth or twentieth day. At an earlier period the congestion round the glands would be more intense; while at a later period the summits of the prominences would become disorganized. Minute yellow sloughs subsequently form, which, becoming detached, leave an ulcer previously occupied by the gland. The mucous membrane around participates in the process. It is red, tumid, and covered with an aphthous layer of lymph, to the extent of one or two inches

around, with three or four solitary glands prominent in the midst. The ulcers which form result from sloughs rather than from ulceration; and the disease still remains *not severe*, as regards the amount of tissue involved; but *as to duration*, the illness may be prolonged and tedious; and the solitary gland cavities may enlarge very much—so large as a horse-bean. (2.) In more severe forms a greater variety and extent of tissue is involved; and especially of the tubular glands. There is great redness, tumefaction, and softening of tissue; and along with the change of color and of texture the secretions are greatly altered. The clear mucus is highly charged with albumen, and subsequently with blood.*

These changes occupy principally the prominences of the transverse rugæ. The exudation on the free surface in the recent condition may form a thin opaque membrane three or four lines thick. It is homogeneous, and of considerable consistence, so that it may be detached, and raised in flakes from the subjacent surface. With the exception of increased vascularity, the subjacent mucous surface appears still unchanged. A microscopic examination of this exudation shows that it varies with the severity of the case to some extent. In mild cases it is simply particles of epithelium mixed with amorphous granules. In more severe forms the exudation consists of fine germs with nuclei, mixed with elongated cell-forms (connective tissue cells); and the examination of carefully prepared sections shows that such exudation mainly commences in the tubular glands, and by proliferation subsequently spreads over the mucous surface as described. In Dr. Clouston's cases examination of the fibrinous layer (over the small intestine), in the fresh state, showed its structure to consist "of nucleated cells like pus-cells, puriform cells with nucleus, and a fibrinous material between them." The bloodvessels underneath the "fibrous layer were enlarged and very tortuous, and on the free surface of the mucous membrane, between the villi, they could be seen torn and open-mouthed." (*Med. Times*, l. c.)

Exuviae, or casts of the intestine, may now be thrown off in large masses or shreds, leaving a raw-looking vascular surface underneath; and in some respects this process and these casts are analogous to similar phenomena in *croup*, *diphtheria*, *dysmenorrhœa*, and to *typhoid fever*. By carefully examining the evacuations, important information may be obtained as to the nature of the process going on in the intestines. Dr. Goodeve, of Calcutta, has made some valuable observations of this kind. He recommends that the evacuations should be washed with water, so as to get rid of the fecal matters entirely; and so leave the sediment, which is the product of the colonic disease, free from bile, fecal matter, and

* Some account of the chemistry of the stools in dysentery has been given by Oesterlen, who describes an excessive elimination of water and of albumen. It is very desirable that this observation should be verified in India. The stools should be collected free from urine; and the albumen should be estimated separately from any insoluble sediment. If in severe cases two and a half ounces of albumen are passed in twenty-four hours by stool, it is impossible to over-estimate the importance of such an occurrence.

offensive smell. [Dr. Chuckerbutty* found the sediment, after washing by the plan of Goodeve, to consist of a certain mucus,—ropy, gelatinous, branny, or thready; shreddy, or granular lymph; laudable, ichorous, or scrofulous pus; fæces in various degrees of consistence; and sloughs.] He observed that patches of membrane, half an inch or an inch or more in size, are cast off as sloughs. These exuviæ are thin, membranous, and sometimes infiltrated with pus; or they are thick and of a yellowish-brown color. It is not till after the *eighth* or *twelfth* day of the disease that such sloughs are cast off. In these respects they may be considered similar to those cast off from Peyer's patches in typhoid fever. After these shreds are cast off the symptoms diminish, and the patient often gets well rapidly.

[The conditions indicated by the various kind of sloughs are, according to Dr. Chuckerbutty, as follows:

CHARACTER OF SLOUGHS.	CONDITIONS DENOTED.
Ecchymosed,	Abraded or minute ulcers; intestinal apoplexy.
Compact gray or light-yellow,	Acute phlegmonous dysentery.
Thick pus—infiltrated mucus,	Erysipelatous dysentery.
Ragged,	Gangrene
Dark olive,	Secondary gangrene of mucous coat.
Thin black, plain, or tubular,	Primary gangrene of mucous coat.
Shreddy,	Gangrene in either mucous or cellular coat.
Molecular, or putrilage,	Disintegration of tissue.
Flaky epithelial,	Commencing gangrene of mucous membrane.
Shaggy,	Violent inflammatory action.
Free filamentous (simple),	Primary gangrene in submucous tissue.
“ (pus-infiltrated),	Submucous cellulitis.
Ring-shaped,	Ring-shaped ulceration in mucous folds.
Discoid,	Circular ulcers in ecchymosed patches.]

During the shedding of the shreds the patients are much griped, and they pass with straining the sanguinolent masses, or slimy mucus in small quantities, and generally without fecal matter, fifteen or even twenty times a day. Then a period of cure and improvement supervenes, with diminution of the stools or of the fecal discharges,—not simply by resolution, but a termination by elimination of the specific sloughs or lesions which have formed in the course of the disease. In this respect the phenomena seem analogous to what occurs in typhoid fever. When these membranous flakes are not shed, but retained and ultimately separated in large pieces, there is considerable danger attending the process. Discharges of blood and fatal hemorrhages may ensue. Morehead records eight cases of this kind in India, and four of them were fatal, of whom one died from hemorrhage. To account for this hemorrhage, it has been observed that changes go on between the intertubular connective tissue and the substance of this exudation,

* [Cases Illustrative of the Pathology of Dysentery, with Remarks. By S. G. Chuckerbutty, Calcutta, 1865.]

which tend to its organization or supply with blood, and subsequently to its destruction, separation, or removal by ulceration. In vertical sections, down to and through the mucous membrane, I have seen fine bloodvessels, in loops and bulbous cœcal ends, shooting upwards beyond the mucous surface into the exudation; and when such exudation was forcibly removed from off the mucous surface, the membrane on which it lay was found to be highly vascular, and numerous minute ruptured vessels showed their torn mouths, by minute points of exuding blood. Dr. Morehead records a similar observation as regards the connection of the exudation with the subjacent mucous tissue, "through the medium of what appeared to be small capillary vessels, the mucous membrane beneath being vascular" (*Researches on Disease in India*, p. 241). In a case that proved fatal at the end of six weeks, and in which the gut was so friable that almost the least current of water tore it up, Dr. Clouston noticed that "in the rectum blood-clots projected from the open mouths of arteries."

When such a state of vascular action in the mucous membrane and exudation has existed for a lengthened period, the tissue of the gut becomes greatly thickened, and at the same time less coherent. These thickened portions grow luxuriantly, just as isolated patches in a field of green corn grow more luxuriantly than others, being supplied with a greater amount of nutritive material, as the thickened patches of mucous membrane are supplied with more enlarged, more tortuous, and more extensively distributed loops of bloodvessels. These thickened masses of dysenteric exudation continue to fungate and grow from a hardened base, from which the numerous bloodvessels pass into the growing masses. In this way the warty condition of the mucous membrane in chronic cases undoubtedly results. The new material evinces a disposition to contract. The calibre or bore of the gut gradually diminishes, and its texture becomes so brittle that slight force in pulling up a piece of such intestine out of its place will readily cause it to *break* asunder.

As to ulceration, it may be readily understood, with such varied lesions, that the formation of ulcers does not take place in any uniform mode; and the following statement is given as a summary of the processes from which ulceration may proceed: (1.) It may occur after intumescence, softening, and simple ulceration of one or of several lenticular solitary glands. (2.) After intumescence, softening, and sphacelus of many solitary lenticular glands and the intervening tissue in one mass. (3.) After softening of the tubular structure and the detachment of sloughs, ulceration follows the intumescence and proliferation of growths from the tubes which cover the surface as a "croupous," "catarrhal," or "diphtheritic" exudation, and to which the name of "aphthous erosions" has been applied. (4.) After submucous inflammation and new growths, with fibrinous and mucinous effusion. (5.) After intertubular inflammation, and after inflammation surrounding the base of inflamed glands. (6.) After the formation of submucous abscess. (7.) By changes of an ulcerative nature, commencing in the vascular exudation itself, as in scorbutic cases.

The *circular* ulcers, for the most part, originate in the solitary glands (PARKES, BALLY), or in circular patches of tubes (MOREHEAD), similarly to the stomach ulceration, as described by Drs. Handfield Jones and Brinton; or such circular ulcers may result from both, as when a solitary gland is destroyed it carries with its destruction some of the adjacent tubes. In such cases the colon presents prominent little masses, about the size of a pea, which burst readily on pressure, and give forth fluid contents like pus. Such abscesses may open spontaneously upon the mucous surface through the short canal leading from the vesicular gland (now an abscess), imbedded in the submucous tissue, and between the tubular glands. They undermine the tubular gland-substance, and carry off shreds or patches of the surrounding tissue. They may thus be seen in all stages, and sometimes almost symmetrically arranged in a double row along the colon (BLEEKER, MOREHEAD). Many of these little abscess cavities are also formed below patches of thick exudation [HASPEL].

The *transverse* ulcers are due to the transverse arrangement of folds, on which the exudation and textures ulcerate, as already described, and I have known the transverse *rupturing* of very thick exudation mistaken at post-mortem examinations for ulceration, on seeing the raw vascular surface of the tissue exposed below at the bottom of the rent.

Microscopically the exudation in its most recent condition may be seen to be composed of fine germs and nuclei, with elongated nuclear cells. It appears to be chiefly exuded into the follicular and tubular apparatus of the mucous membrane, and gradually accumulating there, is pushed upwards to the mucous surface, which it finally overspreads as a whitish coat, coherent and uniform, susceptible of vascular organization, and tending to ulcerate.

Anatomy of the Tissues in Chronic Dysentery.—In the true chronic form of dysentery the exudation already noticed undergoes various changes. It may be thrown off from the mucous surface altogether, leaving that surface bare and raw-looking, as if ulcerated; but a close inspection will show that the surface is entire and highly vascular. If it is not thrown off, it may undergo a considerable amount of organization; after which it appears that a process of ulceration may be established upon its surface, just as in any other soft tissue. This ulcerative process may extend through the whole exudation, even to the surface of the mucous membrane, which it may penetrate also, and involve the tissues of the intestine in the ulcerative process close to the peritoneal coat. Perforation of the peritoneum is by no means uncommon.

In the chronic forms of dysentery there is a very constant morbid change to be observed, consisting in the deposit of black granular matter on some parts of the mucous membrane. It may be regarded as the result of excessive vascular action, and of subsequent changes in the extravasated blood, elements which mark the site of the melanic spot (pigmentary degeneration; see page 121).

The sigmoid flexure of the colon is perhaps the most frequently and the most extensively diseased, and the lesion is most expressed towards the rectum. In very severe cases the exudation extends

over the whole extent of the mucous surface of the *colon*, which appears covered with black, grumous, carbonized-looking masses, even to the upper part. Ulceration is most frequently seen in the *sigmoid flexure*, destroying at once the exudation and the mucous membrane, so as to expose the muscular tissue of the gut, which is red and irritable. An *appearance* of ulceration often extends in lines across the gut, so as to embrace its whole calibre in some parts. This is sometimes, however, only an appearance of ulceration, caused by the separation of the exudation when it is thick, exposing the highly vascular mucous surface below, which looks raw and ulcerated. When the gut is opened in the usual way after death, and extended on a flat surface, the change from the hitherto curved condition of the intestine is so great as to cause rupture or separation between masses of exudation, especially in places where it is thick; thus giving rise to the appearance noticed, and which has sometimes been described as ulceration. In long-continued chronic cases the rectum is generally studded over with punched-out-looking ulcers with bloodless bases and thin anæmic edges; and the melanotic deposit, already noticed, is here seen in the greatest abundance. Evidence of healed ulcers, with partially renewed mucous tissue covering them, are not uncommon in this locality, their place being indicated by the amount of black matter. The gland-tissue, however, is not reproduced in the cicatrix-substance.

Anatomy of the Morbid Tissues in Complex Cases of Dysentery.—In the class of dysenteric cases which may be called *complex* there are a variety of lesions, the pathological significance of which as to extent, form, origin, and locality, renders the cases of dysentery in which they are found of a very complex kind. The lesions which chiefly tend to render cases of dysentery complex, are,—(1.) Extension of the dysenteric process over the mucous membrane of the small intestine; (2.) Deposits and ulcerations in the glands of Peyer, as well as in the general tubular structure near the ileo-cæcal valve; (3.) Atrophy of the glandular parts of the mucous membrane of the alimentary canal; (4.) Secondary lesions of serous membranes; (5.) Secondary lesions of solid viscera in the cavities of the *abdomen* and *thorax*; (6.) Secondary lesions due to the syphilitic, scorbutic, typhus, or tuberculous states, or to the influence of malaria.

In some rapid and acute cases of *dysentery* (five out of twenty-eight cases, as observed by Dr. Baly) it has been noticed that the process by which the *dysenteric lesions* were developed in the large intestine extended beyond the ileo-cæcal valve, and brought about an action in the small intestine similar to that in the colon. As much as the lower two-thirds of the ileum have been involved in this process, while the upper portion has been found intensely congested. In one case of *dysentery*, Dr. Cheyne says he found an exudation of lymph extending nearly over the whole of the jejunum. If the stomach participates in the disease, the mucous membrane may be merely diffusely inflamed, or of a red or violet color, its surface granulated, and its texture broken by the slightest touch. More commonly the color of the mucous membrane is natural, but on its surface a number of ecchymosed spots, or small ulcers, are seen, with

edges so sharp, clean, and perpendicular, that they appear as if made with a punch. In other cases the tubular glands, as well as the solitary and aggregate glands of Peyer, have shown various stages of morbid action. The absorbent mesenteric glands are rarely affected (BALY); but except in cases of secondary hepatic abscess, they were found enlarged and inflamed in all cases of Indian dysentery (PARKES), and also in the dysentery associated with *scorbutus*.

By far the most common condition, however, in chronic cases of *dysentery* especially, is that which is due to atrophy of the mucous membrane. As an atrophic change, it may be ascribed to the general wasting (marasmic) processes which take place to a great extent throughout the system in cases of *chronic dysentery*. In this complex state the mucous membrane of the *small intestine* appears pale, thin, and worn,—a condition which pervades the greater part of the alimentary canal, and which is especially made manifest in the living as well as in the dead by the condition of the mucous membrane of the mouth. On turning down the lips, the mucous glands are seen distinctly projecting through the thin pale labial and buccal mucous membrane. When such cases are examined after death, the structure of the solitary glands and of Peyer's patches are found to be degenerated and wasted; no gland-cells are to be seen, and their place is supplied by fibroid tissue, with some vascular injection round the reticulated spaces. In other instances a deposit of black pigment surrounds the locality of the glands, which indicates the long-continued process of vascular action previous to their atrophy. Associated with this general atrophic state, some gland-patches may be observed in an apparently opposite state—that is, distended, and sometimes engorged; but, on examination, their contents appear to be undergoing a molecular, melanotic, and generally fatty degeneration, probably preparatory to complete evacuation and destruction of the gland-element. These two apparently opposite conditions, coexisting in the same cases, appear to indicate that the one condition is but the antecedent of the other; and that the atrophy and degeneration is the last result of a series of morbid processes commencing in the engorged gland-cavities.

In parts of the mucous tissue which exhibited the opposite conditions of extreme hypertrophy and extreme atrophy, the specific gravity of the former indicated 1.046, while the thin and wasted part of the intestine indicated a specific gravity of 1.036 to 1.030.

There is now abundance of evidence to show that, in some endemic cases, or in epidemics of *dysentery* in some places, there is a tendency to the secondary affections of organs or parts, during or subsequent to the development of the dysenteric process. Some look upon these secondary processes in relation to the dysentery as in the relation of effect following a cause; or that there is an immediate and direct connection between the primary dysenteric process and the secondary lesion. Such a relationship has not been shown to exist in all cases; and it is more probable that the *dysenteric* process, when it operates on the system during a protracted period, predisposes, as many other morbid states do, to the development of secondary local lesions in distant parts.

The arachnoid, the pleuræ, the pericardium, and the peritoneum have each and all of them in some instances been the seat of opacities or of fluid exudations in dysenteric cases.

Of morbid states of the solid viscera, associated with dysentery, by far the most frequent complication is that with the *kidney* and the *liver*. With regard to the kidneys, their relation to the bowel affection is as yet obscure; but in mild cases, proceeding to a favorable termination, there is no albumen and no casts in the urine. When, on the other hand, the dysentery is severe, it continues some time before exudation appears in the urine, and then its occurrence is preceded and attended by putridity of the copious stools, by *status nervosus*, collapse, and paralytic phenomena. If the renal affection occurs early, so much more severe is the case, and death usually speedily ensues. The kidneys after death are seen to be highly congested, the tubes loaded with exudation, cells, and detritus (Zimmerman, *Syden. Society Year-Book* for 1861).

The association of hepatic disease with *dysentery* would seem to be most frequent in the climate of the East Indies, and in such climates as have a similar influence (MARTIN). In the Bombay Army, out of thirty fatal cases of *dysentery*, twelve were attended with hepatic abscess (MOREHEAD). Dr. Macpherson, Sir James McGrigor, Dr. Parkes, and Mr. Henry Marshall, gave similar statistical results of their experience at Calcutta, Moulmein, and Ceylon. The French surgeons in the province of Oran, in Algeria, state that hepatitis and consequent abscess were frequently coincident with dysentery. Dr. Parkes observes, that if the functional morbid state of the liver is to be judged of by chemical analysis of the secretion of that viscus, the liver is found to be diseased, more or less, in every case of dysentery. Dr. W. J. Moore, Assistant-Surgeon of the Bombay Army, has collected a valuable series of statistics, which sets in a stronger light than hitherto the relation of hepatic lesions to dysentery. The records of five independent observers show lesions of the liver (not being abscesses) in about 57 per cent. of the cases; while the number of cases in which abscess was observed averages about 18 per cent. (Grant's *Annals of Military and Naval Surgery*, vol. i, p. 227). The tendency to hepatic complication was found in Algeria to increase with age, and with the length of service in that country. It appears, however, that hepatic abscess is but rarely associated with dysentery in natives of those warm climates; and amongst British subjects in their native climate it seems equally rare. In the Millbank Prison, "out of many hundred cases, not one has been complicated with hepatic abscess." It does not appear, however, that the influence of the climate of the East alone on Europeans tends to the hepatic complication, for "in the Peninsular Army, under the Duke of Wellington, the spleen, the liver, and the mesentery were generally found diseased in cases of dysentery; so were these viscera in the epidemic dysentery of Ireland" (MARTIN). In the dysentery of the allied armies in the hospitals of Scutari and the Crimea during the late Russian war, hepatic abscess was of rare occurrence. Dr. Budd attempts to explain how hepatic abscess is a consequence of dysentery through the vitiation of the portal blood

from the morbid intestines. But the evidence tends rather to show that the hepatic lesion and the dysentery are each excited by the same cause. If Dr. Budd's theory were correct, we ought to have liver abscess a common occurrence after ulcerations of typhoid fever, and after those of tuberculous lesions of the intestines; but we do not find that such a lesion of the liver is usual in such cases.

Regarding hepatic complication in dysentery, the following conclusions may be stated: (1.) That dysentery, in a great number of cases, more than half, commences and runs its course complicated by obvious functional hepatic disease; (2.) That the hepatic disorders and the dysentery acknowledge a common cause and disease-process; (3.) That about 18 per cent. of the fatal cases of *dysentery* are complicated with hepatic abscess; and about 57 per cent. with hepatic lesions; (4.) That in a few of these cases ulceration of the intestine may be the primary disease, and the source of the hepatic abscess by the phenomena of thrombosis and embolism in connection with the pelvic veins and veins of the mesocolon.

The occurrence of hepatic abscess with dysentery has been generally viewed as a result of phlebitis; but Dr. Parkes, after the most careful observation of such cases, never found the slightest trace of inflammation in the small veins of the intestines, while no direct proof has been advanced of the mediation of the portal blood in the process; and in conclusion, writes Dr. Hensch, "I believe we must give the preference to that view which regards the two diseased processes, dysentery and abscess of the liver, as running their course together, dependent upon one and the same cause; in favor of which view is the circumstance, that in hot climates abscess of the liver very frequently occurs associated with remittent fevers, or consecutive to them, without dissection exhibiting any ulceration of the mucous membrane of the intestines" (*Brit. and For. Med.-Chir. Review*, July, 1854). The comparative frequency of the occurrence of hepatic abscesses may be seen from the following statement:

In *Calcutta* General Hospital they occur at the rate of 13.1 per cent. (MACPHERSON); in the Medical College Hospital, at the rate of 25.9 per cent.; in *Bombay* General Hospital, at the rate of 40 per cent. (MOREHEAD); and in *Madras* Presidency, at the rate of 50.97 (ANNESLEY), 19.35 (PARKES), 17.9 (INNES, at Secunderabad); MACNAMARA, in Madras, 50.9 per cent.; French surgeons in Algeria, 12.7 per cent.; EYRE, of the Madras Fusiliers, 22.8; WARRING, in various localities not stated, 23.5 per cent.; STOVELL, in European General Hospital, Bombay, 19.3 per cent.; LEITH, in Bombay, 8.5 and 15.2 per cent.; and MARSHALL, in Ceylon, 28.8 per cent.

Too much attention and importance seem to have been put upon abscess of the liver *per se*, irrespective of other obviously morbid conditions of that organ—*e. g.*, impaired functions, congestion, enlargement. To regard secondary hepatic abscess as due to absorption of pus or other morbid matter from ulcerating mucous membrane, or to a true phlebitis, is to take too narrow a view of the relation of liver disease to dysentery; for if we are to judge by the condition of the bile alone, the liver is diseased (in function, at

least) in every case of dysentery (PARKES). The contrast of the results given in the above table, with the result of the cases seen in colder climates, is indeed remarkable. Baly's experience yielded him no abscesses of the liver. Finger, of Prague, dissected 231 cases of dysentery between 1846 and 1848, and found no abscess of the liver. Broussais records seventeen dissections of dysentery in the camp during 1805 and 1806, and no abscesses of the liver. Rokitsansky has never found the liver visibly diseased in cases of dysentery; and in China, where dysentery, as a rule, is very fatal to Europeans, the rarity of hepatic abscess is said to have been remarkable (Dr. Wilson in *Records of Hospital Ship "Minden"*).

On the whole, it will be seen that the association of dysentery with hepatic abscess is not equally frequent in all countries, nor in all epidemics. It seems to have been most frequent in the climate of the East Indies, and in the Bombay army especially (MOREHEAD, PARKES). There are some epidemics in Europe in which the hepatic lesion has been observed; *e. g.*, in Dublin, 1818, it was observed in four out of thirty cases (CHEYNE). It would therefore appear that the *poison* which causes dysentery has at some times and places the power of establishing hepatic complications so severe as to lead to abscess; at other times and places it seems to be less virulent.

The spleen and pancreas are sometimes also found diseased; and Mr. Twining notices the former as one of the most fatal complications of dysentery in the East Indies. These viscera are found either enlarged and softened, or enlarged and indurated, the spleen being sometimes the seat of abscess.

Of thoracic viscera, the lungs have sometimes exhibited a great tendency to secondary morbid processes in dysenteric cases. This was especially the case in the dysentery of the allied armies during the late Russian war, where otherwise pulmonic lesions were rare.

The pulmonic lesions associated with the dysenteric process were as follows: (1.) More or less extensive lesion of the bronchial membrane, the finer ramifications of the tubes being filled with frothy mucus and pus-like exudation, and associated with extensive vesicular bronchitis: there were well-marked spots of lobular pneumonia. (2.) Exudations into the pulmonary parenchyma, chiefly in the form of isolated deposits of considerable density, disseminated through the substance of the lungs. These masses passed into a purulent condition, and microscopically they were composed of broken-up cells, granular matter, and pus-elements.

The last class of conditions which render cases of dysentery complex is the alliance of other disease-processes with dysentery. Such cases are generally of a very protracted duration; and the associated morbid lesions are not only complex from the number of morbid processes developed and the organs attacked, but they are complex from the variety of kind, degree, and extent of the coexistent affections. Many disease-processes may be observed to coexist in one patient; and such multiplicity of disease-processes tends greatly to multiply the number of the anatomical local lesions, and thereby still more to complicate the case.

Dysentery "is found to complicate readily in all climates with the prevailing fevers." Within the tropics it is frequently associated with *remittent* and *intermittent fevers*; in the geographical region of *typhus fever* it is a most frequent complication, under various circumstances, and becomes capable of propagation from person to person; and, lastly, it is also occasionally complicated with *scurvy*. When dysentery follows upon, or is associated with, intermittent fever, the spleen will frequently become enlarged, indicated in the outset by general anæmia, or splenic cachexia, with a low asthenic type of dysentery.

The scorbutic complication is developed in cases of *dysentery* when the supply of food has been deficient in fresh vegetables, or when it consists in whole or in the greater part of salted meat. Sir Gilbert Blane asserts that the complication has been known to arise among prisoners of war, living entirely on fresh (animal) diet.

"The most terrible instance of suffering from this cause," writes Sir Ranald Martin, "was that of the European portion of the force employed in Ava during the first Burmese war, where they were for six and a half months fed on salt rations, and where 48 per cent. perished within ten months, principally by dysentery with the scorbutic state." Such disasters have since been equalled, if not surpassed, by the sufferings of our troops in the camp before Sebastopol during the winters of 1854-55, under the influence of exposure, fatigue, and continued rations of salt meat and green coffee.

There is still another light in which the pathology of this disease requires to be studied, namely, in the

Types and Forms of Dysentery.—These have been variously described as (1), the *purely inflammatory, acute, hyperacute, or sthenic* form. In this form, while the phenomena indicate acute and severe inflammatory action, there is no tendency to the great depression of the nervous, circulatory, and muscular functions, which gives a marked character to some of the other types of the disease, such as (2), the *asthenic* forms. In the *asthenic* forms, besides the depression of the functions just noticed, there is much greater tendency in these forms to spread by infection, or under an epidemic influence. These asthenic forms are sometimes described as *adynamic, typhoid, malignant, bilious, intermittent, or remittent*, according as certain phenomena prevail characteristic of these states.

Symptoms of Dysentery.—An ordinary attack generally commences with *diarrhœa*; but in twelve or twenty-four hours disagreeable feelings begin to attend the frequent loose discharges from the bowels. These are irregular pains, commonly called "gripes," along the course of the large intestine, and sometimes described as "shooting," or "cutting." Technically, such symptoms are known as *tormina*. They are momentarily relieved by discharges from the bowels. But after a short time a sense of heat ascends from the rectum, and pain extends to the epigastrium, till the whole abdomen is painful. There is a frequently returning inclination to go to stool: the griping and straining continue without the patient being able to pass anything more than a little bloody mucus. These symptoms, are generally aggravated during the night and early

morning, and they leave behind them the exhausting sensation that there has always remained in the bowel something which has yet to be discharged. This feeling is technically called *tenesmus*, and ultimately becomes the most striking feature in the case. The acute pain in the abdomen, although it may extend to the iliac regions or flanks, generally concentrates itself at last about the rectum.

The discharges from the bowels are at first scanty, consisting of mucus and blood, or bloody slime, as it is sometimes called. As the disease progresses, the evacuations become more copious, tinged with bile, and carrying off shreds of the exudation thrown out on the mucous surface of the intestine. Hardened balls of feces called *scybalæ*, are also sometimes discharged: these, however, are seldom seen in tropical dysentery, and if much feculent matter pass, there is always considerable relief. When the disease is fully established, the discharges exhale an odor different from the smell of feces, and which is almost peculiar to dysentery, and very offensive. It is important to observe the character of the discharges, and especially as to the relative amount of blood, mucus, and shreds of exuvie. If the disease advances, besides the constitutional symptoms becoming aggravated, more blood and mucus appear in the discharges from the intestines, together with shreds or large sloughs of exudation, which are often described as pieces of mucous membrane. In very acute cases, going on rapidly to an unfavorable termination, a great change often takes place in the nature of the stools, which become suddenly copious, serous, of a reddish-brown color, with black spots, attended with a putrid, offensive odor, which pervades the whole house. In the acute dysentery of Lower Bengal the patient is not unfrequently carried off by copious discharges of blood (W. C. MACLEAN). The shreds, however, are not mucous membrane, but like the dysmenorrhœal membrane which forms on the internal surface of the uterus, the dysenteric slough varies in consistence, thickness, and strength. It may be washed perfectly white in water, and its minute histology shows no character of a mucous membrane. The hardened balls of feces are much more rarely seen than they have been described to be. When the skin is dry, and of a pungent heat, the tongue furred, and the thirst urgent, the urine scanty and high-colored, and the pulse increasing in frequency—these are symptoms of increasing danger in dysentery. Throughout the disease there is febrile distress, the nights are passed without sleep, or, when it is obtained, it is in short periods, dreamy and disturbed; and when the patient awakes he is unrefreshed, and his spirits low and desponding. In the majority of cases the disease takes a favorable turn between the sixth and tenth days; the symptoms are then mitigated, the pain ceases, the number of stools diminish, and the flow of urine is restored. On the contrary, if it terminates fatally in this stage, hiccough, vomiting, a small and rapid pulse, and pale sharp features, denote the approach of death. The intellect, however, is perfect, and the patient, often deploring the fate which he sees inevitably to await him, dies after a short agony. In the dysentery described by Dr.

Clouston some of the patients had ordinary diarrhœa (diarrhœa of irritation?), from periods varying from two or three hours up to twenty-four hours, before blood appeared in the stools. In some cases there was great pain in the abdomen for twenty-four hours before the diarrhœa set in (evidence of irritation?). In other cases there was scarcely any pain at any period of the disease.

Dr. Clouston distinguished two classes of cases. "In the first the patient had two or three loose stools, or perhaps had no ordinary stools at all, but at once began to pass glairy mucus, mixed with blood, in small quantities at a time, from the bowel. He had no pain, no fever, no want of appetite, and he refused to believe he was ill. This would continue for a day or two, and then the blood would increase in quantity, and the stools would become more frequent. Pain would begin to be felt in the region of the rectum, and the pulse would mount up by ten or twelve beats. For days the patient would be at stool every hour or two, and of course would become weaker. His tongue was then seen to be coated with a dirty yellowish-white fur; but the appetite for such forms of nourishment as milk, strong beef tea, calves' foot jelly made with wine, was still good. Solid food was not relished. These stools would then be seen to be coated with a semi-fibrinous semi-purulent-looking membrane. The tongue would then become clean, and glazed, and beef-steaky; the evacuations became feculent, mixed with pus, the latter element becoming gradually less as the patient advanced in his slow convalescence.

"In the second class of cases the patient had from the first great pain in the abdomen, of a griping kind, a hot skin, and a pulse over 100; the dejections were copious, and frequent, and watery, while they were largely mixed with blood. In many cases there was sickness; in all, loss of appetite. After some days the tongue and mouth would become dry, and parched, and black; the features pinched; the pulse small and quick; and death soon ensued. In some cases the stools would, after a time, become membranous and shreddy, and then purulent, till the patient was more gradually weakened and exhausted. One such case lived six weeks, another two months. In one only of this class of cases (out of seventeen) did the patient recover.

"All the cases had the following features in common: Bloody stools at first, tending to become purulent; intense fetor of the evacuations during the whole of the disease; no scybala, and great thirst" (*Med. Times and Gazette*, June 10, 1865). These were cases of dysentery caused by the poison of animal effluvia from decomposing human excreta (undiluted) acting on constitutions in which vegetable diet seems to have been deficient (scorbutic), and in whom the nervous power was below par.

If the disease proves fatal in the chronic form, the patient generally becomes rapidly altered and prostrated by his sufferings, is strikingly emaciated, and often earnestly prays to be relieved from a life disgusting to himself and entirely despaired of by others. Death begins at the heart.

The patient, on the other hand, may in a few rare instances re-

cover; the local symptoms gradually yield, till his health and strength are ultimately restored, in a moderate degree. Convalescence is slow, rarely complete; and there is perhaps no disease which makes so persistent and pernicious an impression on the human constitution as dysentery.

Causes and Modes of Propagation.—It may be stated, as a general proposition, that there is no country where paludal fever exists in which dysentery is not an endemic and prevailing disease. In the East and West Indies, in China, the Ionian Islands, Gibraltar, Malta, the Canadas, Holland, the coasts of Africa, as well as in many different parts of France, of the Peninsula, of the continent of America, and of the eastern parts of Great Britain, the prevalence of intermittent or remittent fevers and of dysentery is notorious.

This connection is so intimate that a given number of persons being exposed to the action of paludal miasmata—as, for example, a boat's crew sent ashore in a tropical climate—the probabilities are, that of the men returning on board, part will be seized with dysentery and part with remittent fever.

Paludal fever and dysentery, moreover, are not only conjoined in locality, but they often coexist, precede, or follow each other in the same individual, so that the fever frequently ends in dysentery, and the dysentery in remittent fever. This proof of the common nature of these diseases is corroborated by every writer of any celebrity, and more especially by those who have detailed the diseases of our armies. But dysentery also prevails where there is no other evidence of the presence of malaria. Nevertheless, the evidence in favor of malaria being the common, though probably not the sole cause of dysentery, appears to be much the stronger. It seems also determined that dysentery prevails generally in the inverse ratio of the intensity of paludal fever. In Jamaica, for example, when the white troops suffered in the large proportion of 91 per cent. annually from paludal fevers, the cases of dysentery were to those of fever as one to nine; while in the Madras presidency, when the troops suffered from fever in the much less ratio of only 30.25 per cent. annually, the cases of dysentery were to those of fever as forty-seven of the former to thirty of the latter. It appears that dysentery is less common in the hotter than in the colder months, or arises under circumstances less favorable to vegetable decomposition. Thus in India and China it is from the middle of November to the latter end of February, or when remittent fever changes into intermittent, that dysentery greatly prevails. It seems to be recognized by those most competent to judge, that there is a directly exciting action of malaria, quite apart from that indirect action of undermining the general health, the importance of which as a predisposing cause cannot be over-estimated (GRANT, W. C. MACLEAN).

Our knowledge of the predisposing causes is derived from what principally occurs in the military and naval service; and from the sufferings of the troops we learn that exposure to the night air, to wet, or to fatigue, together with the intemperance and often improper diet incident to the life of a soldier, especially on active ser-

vice in the field, have at all times been found to be conditions powerfully predisposing to dysentery.

The effects of *salt* diet in the production of dysentery being less known than the other predisposing causes, it may be as well to state that, by an experience of twenty years in the West Indies, it has been determined that in the Windward and Leeward Command, when the rations issued to the troops consisted of salt provisions five days in the week, the mortality from diseases of the stomach and bowels among the officers was as two to four per cent., while that among the soldiers was as 20.7, or a tenfold ratio. On the contrary, in Jamaica, when salt provisions were issued to the troops only two days in the week, the mortality from the same diseases approximated so nearly between these two ranks as to be almost an equality. And corresponding facts to these have been observed in Gibraltar, on the coast of Africa, and at St. Helena. The Sierra Leone Commissioners on the western coast of Africa, who investigated this subject on the spot, were of opinion that the large proportion of salt rations mainly contributed to the sickness and mortality from diseases of the stomach and bowels in the form of dysentery and diarrhœa; and the following statement, given by the late Sir Alexander Tulloch in his Statistical Reports (page 11) on the sanitary condition of the troops in the West Command, shows the marked reduction which took place in the deaths from this class of diseases subsequent to the introduction of fresh meat diet; the mortality being reduced to a *tenth* part of its former ratio:

PREVIOUS TO ALTERATIONS IN RATIONS.						SUBSEQUENT TO ALTERATIONS IN RATIONS.					
Year.	Mean Strength.	Dysentery and Diarrhœa chiefly.		Ratio per 1000 of Mean Strength.		Year.	Mean Strength.	Dysentery and Diarrhœa chiefly.		Ratio per 1000 of Mean Strength.	
		Admitted.	Died.	Admitted.	Died.			Admitted.	Died.	Admitted.	Died.
1825	571	235	82	411	56	1828	232	189	1	600	5 $\frac{1}{10}$
1826	471	256	26	543	56	1829	114	50	—	439	
1827	845	209	13	606	38	1830	42	22	1	524	
						to 1836					
Total,	1387	700	71	Average. 505	Aver. 51	Total,	388	211	2	Average. 543	Aver. 5 $\frac{1}{10}$

In the navy the same effects of ill-regulated diet have been observed, and the good effects of a change. "In 1797," says Dr. Wilson, "the victualling (of the navy) was changed, greatly improved, and consequently immediate to the change the health of the seamen improved strikingly. *Scurvy, typhoid fever, dysentery, and ulcer*, which, up to the period of the change, had produced great havoc, became comparatively rare in occurrence and light in impression," and, it may now be added, are hardly known except by name.*

* As Dr. Christian justly observes, the salt meat of military and naval rations is not the same as the salt meat of civil life. The former is highly salted, in order to

An insufficient diet was the main predisposing cause of the dysentery which prevailed in London at the Penitentiary, Millbank, shortly after its completion. This prison is built on a marsh below the level of the Thames at high-water, the river being banked out by a narrow causeway. As long as the prisoners were allowed a full and ample diet, they appear to have resisted the action of the paludal poison, and to have enjoyed good health. No sooner, however, was the quantity and quality of their dietary lowered than dysentery of a very fatal character broke out, and made it necessary to clear that establishment for a time of all its inmates. In the dysentery so well described by Dr. Clouston (already frequently referred to), the predisposing cause seems traceable to a diet deficient in fresh vegetable food, in constitutions of feeble nervous power, and aged, weak, and paralyzed persons, exposed to the poison of animal effluvia, in the form of decomposing and undiluted human excreta, as an exciting cause of the disease. The morbid appearances were characteristic of the scorbutic form of dysentery; and the premonitory symptoms were similar to those described by Dr. Barker (and referred to at page 381 of this volume) as peculiar to poisoning by sewage gases. Such cases were not contagious.

From the MS. notes of Dr. Alexander Grant regarding dysentery in India, to which I am kindly permitted to refer, I find that he, too, is of opinion that sewage miasm (animal effluvia poison?) may be a direct exciting cause of diarrhœa and of dysentery, constituting one form of the disease.

In cachectic states, whether from malaria, syphilis, scorbutus, the exhaustive effects of heat long continued have a markedly predisposing influence in favoring the production of dysentery; and in the treatment of the disease the existence of any such cachexia must be inquired into, and if possible counteracted.

There are few facts to enable us to determine the proportions in which the different ages suffer from dysentery, but the returns of the troops from the Mauritius show that the mortality from this disease falls principally on soldiers advanced in life (TULLOCH).

FORCES IN THE MAURITIUS.	AGE.			
	18 to 24.	25 to 33.	33 to 40.	40 to 50.
Aggregate strength of seven years, . . .	3892	5361	1215	300
Died of Dysentery,	26	63	24	8
Ratio per 1000 of mean strength,	6.7	11.8	19.7	36.6

Besides unwholesome solid food, water of an impure kind and from an impure source favors the development of dysentery.

keep for two or more years in every climate. Its nutritive value is thus greatly overrated, and its nutritive constituents are still further diminished by the process of washing out in water before it can be eaten. Thus, besides the irritant effects of the salt diet in producing dysentery, another element exists as a cause of disease—namely, the insufficient nutrition which the salt ration diet is able to impart.

Drained from swamps, and used for drinking and cooking purposes, as it was on the Chinese coasts, it exerted a marked injurious influence both in exciting and in maintaining the disease. In connection with impure water, the reader is requested to refer to what is said afterwards under the head of "Parasitic Diseases;" and especially under "*Distoma hæmatobium*."

Many other predisposing causes favor the development or propagation of the disease, especially amongst soldiers in active service—namely, long marches in hot weather, bivouacking at night in the open air (often extremely cold both absolutely and relatively to the day), want of sufficient clothes and bedding, may be mentioned as the chief.

It does not seem to be so clearly understood as it ought to be, that dysentery is contagious, or rather that it is capable of being propagated from person to person. Being a frequent complication or concomitant of contagious fevers, it has been believed to inherit similar contagious properties. In the severe form of dysentery, for which the old Infantry Barracks of Secunderabad, in the Deccan, have long been notorious, it has been observed that men, laboring under other diseases, who happened to be exposed to the putrid effluvia of the excretions of dysenteric patients, were often severely affected by the disease (W. C. MACLEAN). There is, therefore, good reason to believe that the exuvie of dysenteric patients, as passed by stool, may, like those of typhoid fever, propagate the disease; and the observations of Budd and Goodeve give support to this view.

Prognosis.—The prognosis in dysentery depends much on the country in which the disease occurs, and on the combination of circumstances predisposing to the disease, not less than on the form or type which the disease may assume. In hot climates it is calculated that the deaths vary from one in nine to one in twenty; and on actual service the chances of recovery are much diminished. In all returns, however, the total deaths recorded give a faint idea and inaccurate representation of the real mortality resulting from dysentery. If it were possible to trace out the men who were invalided from the army and navy services from the effects of this disease, it would be found that the mortality is very much greater than is represented by tabular returns. It is a malady which, once fairly engrafted on the system, never leaves it till life itself becomes extinct (BRYSON, and others). It is sometimes also insidious in its mode of attack and progress; and there is such a desire, on the part of soldiers especially, to avoid the restraints of hospitals, that the disease is sometimes beyond the power of medicine before coming under treatment, especially in tropical commands (TULLOCH). In the cases described by Dr. Clouston as those of the second class (see page 557, *ante*), the chances of recovery are hopeless from the first, and all complications with scorbutus are very unfavorable. There may be diseases of a more rapidly fatal character, but there are few which entail so great an amount of suffering. When once the disease has passed into the chronic form, it slowly, but not the less surely, continues, by a most loathsome process, to exhaust the vital energies, until death relieves the patient of an existence rendered almost in-

tolerable by pain, debility, and the offensive nature of the discharges (BRYSON).

Diagnosis.—It is difficult, perhaps impossible, in the first stage, to distinguish dysentery from diarrhoea; but the blood, the number of the stools, and small quantity of fecal matter passed, will, in times when dysentery is prevalent, indicate the true nature of the disease.

Treatment.—After what has been written regarding the nature and the causes of dysentery, it is the obvious duty of the physician to direct his attention, in the first instance, to the *prevention* of the disease. He must inquire especially as to the conditions of the diet, that it be sufficient as to its animal and vegetable elements, and of good quality. Next, he ought to insure the means of detecting the disease early—for time is of the greatest importance in its cure and prognosis—and especially by removing the patient, if possible, from the sphere of action of any of those predisposing or exciting causes as have been noticed; and also to see that his surroundings are free of all those circumstances which co-operate in aggravating the disease, the chief of which are overcrowding, bad ventilation, bad food, exposure, intemperance. With regard to medicinal agents: "He who would treat this disease with success," writes Sir Ranald Martin, "while he shuns exclusive means, must assign to each remedy its proper value. Bloodletting, sudorifics, and purgatives, constitute the most universal remedies, and in simple uncomplicated dysenteries they will prove all-sufficient. But when the abdomen is tumid, and there is pain in the liver, or in any other region, while the nature of the discharges indicates advancing inflammation, calomel, conjoined with sudorifics, and repeated to meet the occasion, will powerfully aid the curative effect through its influence on the depurative functions—on the circulation, by unloading, jointly with purgatives, the gorged vessels of the abdominal organs—on the blood and on secretions generally—and on the very sudorific function which we wish to excite. While calomel is a most powerful agent when used judiciously, as an aid to bloodletting, pushing it to the extent of ptyalism is by no means to be recommended; nor should mercury in any shape be used in *adynamic* forms of the disease, in scorbutic dysentery, or in poisoning by animal effluvia, in the splenic cachexia, nor in states of anæmia, for in all these conditions of the system its actions are most injurious" (see footnote by Dr. Maclean).

Ipecacuanha was formerly, and still is, much in vogue as a remedy in the treatment of dysentery; but although highly useful in some conditions, it is not to be regarded as a specific in all forms of the disease. There is no exclusive plan of treatment applicable in all cases. Admitting, therefore, the necessity of occasionally employing general and local bleeding, and also calomel as a laxative, in cases of hepatic complications, we have beyond this only the general principles to guide us of allaying irritation, and of controlling, if possible, the diarrhoea; and the best general rules that we possess are those recommended by Sir James McGrigor to be adopted in the army (acknowledged by him to be derived from Dr. Somers); those given by Sir Ranald Martin, already detailed; and those by Professor Maclean, about to be noticed.

"In acute cases," says Sir James McGrigor, "we commenced by copious venesection, and immediately afterwards gave twelve grains of compound ipecacuanha powder every hour, which was repeated three times, with plenty of barley-water, and profuse sweating was encouraged for six or eight hours. A pill of three grains of calomel and one of opium was administered every second night, and in the intervening day, two drachms of sulphate of magnesia dissolved in a quart of light broth. The venesection was repeated, while the strength and pulse permitted it, until the stools were free, or nearly free, from blood. Dover's powder as a sudorific was always given after bloodletting.

"In cases where the pains were excruciating, and attended with tenesmus, the warm bath gave instantaneous relief. This plan being steadily persevered in for a few days, the inflammatory diathesis of the intestinal canal, which had excited symptomatic fever throughout the general system, was found gradually to be relieved, and paved the way for returning health. In this stage gentle tonics, with light nourishing diet cautiously taken, and at first given in moderate proportions, were administered with the happiest effects.

"The disease was not unfrequently cut short by this method. If, however, the disease became chronic, a different mode of treatment was pursued, and not unsuccessfully, if the disease had not been of long duration, the intestinal canal not much disorganized, or not complicated with other diseases.

"The first indication in this chronic state was to relieve the tenesmus and procure easy stools, and with this view ipecacuanha was given, sometimes with calomel, sometimes without it. The neutral salts were given, or castor oil, jalap, and various other medicines of the same class. The second indication was to relieve the number of the stools, and to restore tone to the alimentary canal. With this view Dover's powder, the compound powder of chalk with opium, astringents, and demulcents, with aromatics, were given, occasionally interspersing laxatives, and obviating particular symptoms as they occurred. Lastly, an infusion of bitters was given, to restore tone to the relaxed intestine."

In addition to these remedies, Sir James McGrigor states that the *balsam of copaiba*, an *infusion of calumba*, *hæmatoxylon*, *kino*, and *catechu*, assisted by *opium* occasionally, gave much relief, and also the administration of a variety of enemata, and especially one of a strong solution of *superacetate of lead*; while in cases of liver affection, he adds "that friction of the abdomen with mercurial ointment gave the least irritation, and at the same time produced less debility."

Such is the statement of the practice pursued in dysentery during the Peninsular war, on a scale whose magnitude has seldom been surpassed even in modern times. If, however, we look to the returns, we find it highly probable that not more than two out of three of those attacked ultimately recovered.

The bark of the root of *calotropis gigantea* (or *mular*) has been recently used in India, and found to be an excellent substitute for *ipecacuanha*. It is used in doses of a scruple to a drachm, is a relia-

ble cholagogue, and sedative to the muscular fibres of the intestine, rapidly allaying pain, tenesmus, and irritation (Durant, *Ind. Med. Jour.*, May, 1866).

In general, the dysenteric patient is not admitted into the hospitals of our large towns until the disease has passed into the second stage; and there is no class of disease which then offers so few chances of recovery. On the Continent the neutral salts and mild purgative medicines are highly spoken of; but it is difficult to understand how these substances, having no specific power over the disease, can be beneficial in a highly ulcerated state of the intestine. Of all purgatives, however, two ounces of an infusion of *ipecacuanha* (in the proportion of one drachm to a pound of boiling water), combined with five to ten drops of the *tincture of opium*, and given every six or eight hours, appears to be the best; but the disease, though mitigated, is seldom cured by these means. Vegetable tonics, containing *tannin*, as *kino*, *hæmatoxylon*, or *catechu*, however prepared or combined, give temporary relief, but are ultimately inefficient. When the disease has fairly gained the ascendant, it does not appear that one remedy is better than another. Dr. Bryson writes that he has seen all the astringents, both mineral and vegetable, mercury, both internally and externally, with many other medicines, tried without any benefit; but there were some means which were useful in relieving the more urgent and distressing symptoms, and, as it were, in smoothing the path to the grave. Amongst these he mentions a *well-regulated farinaceous diet*, *opium suppositories*, *anodynes*, *astringent injections*, minute doses of *calomel* in combination with *opium*, *cascarilla*, *resinous astringents*, and the application of *leeches to the rectum* when tenesmus was distressing, or over the course of the colon when there was deep-seated pain. An injection of warm starch (two ounces), with laudanum in it, will often give great relief. As much nourishment should be given in a liquid form as the patient can be got to take. Milk boiled with flour should be taken as often as possible, night and day. It should be taken cold, even with ice, and in small quantities at a time; and small pieces of ice not only allays sickness and nausea, but seems to soothe the irritability of the intestines. Strong beef tea, and Liebig's extract of flesh are most useful.

The value of a change of climate, as a curative measure, is forcibly illustrated by Dr. Bryson. He says that the crews of vessels improved in health almost immediately after quitting the station where dysentery prevailed.*

Where sewage is applied to the soil by surface irrigation, it ought to be diluted largely with water, and deodorized by *carbolic acid*.

* My friend and colleague, Professor Maclean, writes me the following note on the treatment of dysentery, the result of his extensive experience in India and China: "The first thing to bear in mind in the treatment of tropical dysentery is, that the appearance of strength in the patient, given by the acuteness of the symptoms, is delusive. Under the use of strong antiphlogistic treatment the strength of the patient is apt to fail suddenly, and this is often the case even when the treatment has been more conservative in its character. It was once the custom in India to deplete freely in this disease, either by a general bleeding or by the repeated application of leeches; but the most judicious and successful practitioners in India rarely bleed now, even in

[CHRONIC CAMP DYSENTERY.]

(DR. CLYMER.)

History.—The extreme frequency and mortality of this disorder in the armies during the late civil war give it interest and importance to the American physician. More than one-fourth of all the cases of disease

the most sthenic forms of the disease, and confine the use of leeches within the narrowest limits.

Mercury.—It is certain, too, that mercury is yearly less and less used in India than it was, and there is much evidence to show that a corresponding reduction in the mortality of the disease has been the result. The objections to its use are numerous,—it entails great suffering on the patient, if pushed to pyralism, aggravating his miseries, and too often permanently injuring his constitution; it has no specific action on the disease, and its cholagogue effects can be attained by remedies which are not open to such objections as can be brought against mercury. (With regard to its cholagogue effects, grave doubts are thrown by the experiments of Dr. George Scott, referred to at the footnote of page 160 by the author.) In sloughing dysentery it is followed by the worst results; and the observations of clinical observers in India have shown that individuals under the influence of mercury are not only not exempt from attacks of the disease, but are peculiarly prone to be affected by it. This is the case in a very marked degree in Asiatics (MORRHEAD and MACLEAN).

Ipecacuanha.—This remedy has long been used in South America in the cure of dysentery,—whence, indeed, it came. It was much used in India until the mercurial notions of James Johnston prevailed. It was again used by Dr. Twining, of Bengal, by whom it was strongly recommended, and also by Dr. Mortimer, of Madras. Twining combined it with blue pill and gentian, and used it chiefly in small and oft-repeated doses. In South America the practice has always been to administer an infusion of the bruised root,—3ij being infused over night in 3iv of water, and given early in the morning. In Peru it is given in doses of ʒss. to ʒj of the powdered root in a little syrup and water. This practice of giving ipecacuanha in large doses has lately been revived in India with encouraging success, and, I believe, the greatest number of cures. It appears to act on the portal capillaries, and on those of the mucous membrane of the bowels, and to determine powerfully to the skin. *It is usually given in doses of half a drachm or a drachm, either in pills or bolus, or suspended in mucilage, according to the fancy of the patient. It is advisable to give an opiate half an hour before, and to withhold all drink for some hours.* Unless there be hepatic complication, it seldom happens that much vomiting is caused by these large doses; on the contrary, they are often tolerated when smaller doses are rejected. The dose should be repeated in about six hours. A sufficient interval should be allowed to intervene between the doses of ipecacuanha to admit of the patient being sustained by nourishment adapted to the stage of the disease. I need hardly add a caution not to press the remedy too far.

Dr. Cornish, of the Madras army, has shown from official documents that the mortality from acute dysentery in Southern India under mercurial treatment was 7.1 per cent. Since the general introduction of ipecacuanha in full doses it has fallen to 1.3. Dr. Ewart, of Bengal, has shown that equally good results have followed the same system in that Presidency. During the forty-two years from 1812 to 1853-54 the mortality among European troops in the Bengal Presidency amounted to 88.2 in the thousand. But during 1860, when large doses of ipecacuanha were administered, the mortality was only 28.87 in the thousand.

Great credit is due to Mr. Docker, of the 7th Royal Fusiliers, for recalling practitioners to the use of this invaluable remedy (*vide Grant's Annals of Military and Naval Surgery*, vol. i).

In no disease is early treatment more necessary than in dysentery, and I believe that, if conducted as above, except in the malignant and 'putrid' forms, we may look for good results in a large proportion of cases. Turpentine epithems and fomentations should be diligently used, and the patient's strength should be supported by nourishment of a bland kind, suited, in degrees of nutritive value, to the stage of the disease.

In the scorbutic form we have a valuable remedy in the *Bael fruit*, when procurable. This fruit contains a large quantity of *tannin*, with vegetable mucus, a bitter principle, and a vegetable acid. It is much used in Bengal; and in the

reported during the first two years of the war—1861-62, 1862-63—was of the several forms of dysentery and diarrhœa; the annual number of cases for the whole army being greater than three-fourths of the mean strength—the ratio of cases being 765 per 1000 of mean strength during the first year, and 852 per 1000 for the second. The mortality was 4.10 per 1000 of strength the first year, and 16.8 the second; for the two years 12.36. After camp fever, they were the chief cause of death by disease. The total number of cases reported during the first year was 215,214 and 1194 deaths; during the second year, 510,461 cases, and 10,366 deaths; total, 725,675 cases and 11,560 deaths. Of acute dysentery and diarrhœa, 196,788 cases and 574 deaths were reported in the first year, and 438,631 cases and 1792 deaths in the second year; the death-rate being 1 to 331 cases in the first year, and 1 to 245 in the second. Of chronic dysentery and diarrhœa, there were 18,426 cases and 620 deaths in the first year; and 71,830 cases and 8574 deaths in the second year; the death-rate rising from 1 in every 30 cases for the first year to 1 in every 8 cases in the second year.* The official statistics of the last two years of the war have not yet been published, but it is known that the prevalence and mortality of the disorder were not lessened.

In the Confederate army diarrhœa and dysentery made 26.7 per cent. of all cases entered upon the field reports, and 19.3 per cent. of those admitted into the general hospitals during the years 1861 and 1862. The exact rate of mortality cannot be ascertained, as the deaths entered upon the field and hospital reports do not represent the total caused by diarrhœa and dysentery, for a large number suffering from the severer forms were either discharged or sent home on furlough, and subsequently died (J. JONES, *U. S. Sanitary Commission Medical Memoirs*, 1868). In the Southern military prisons camp dysentery was very prevalent, and with very great mortality. From a Report made to the Surgeon-General of the Confederate army by Dr. Joseph Jones, it appears that in the prison at Andersonville, Ga., during six months from March to August inclusive, 1864, there were 4529 cases of camp dysentery among less than 40,000 U. S. prisoners confined there. Later, the mortality much increased.

The acute camp dysentery of European armies never prevailed epidemically in the American armies during the late war.

Nature and Causes.—Chronic Camp Dysentery, as it prevailed in the United States armies, was not produced by any one condition; "no specific causative momentum," or epidemic influence, being evident. It was brought on by the long-continued and co-operative action of certain intelligible influences, chief amongst which were the scorbutic taint from defective diet, paludal toxæmia, the filth and overcrowding of camps and barracks, excessive and prolonged heat, and physical fatigue during active campaigns. All these ætic influences, continuous and long-acting, vitiated the proper nutrition of the body, established a condition of slow and persisting semi-starvation—an autophagic state, the body feeding on itself,—death resulting from extreme debility and exhaustion, except in exceptional cases, where some acute intercurrent affection killed the patient. The influence of the scorbutic taint was shown not only by the increased frequency of dysenteric disorders whenever supplies of fresh

scorbutic form I have seen it successful when all other measures have failed (see note on Bael fruit, by Dr. Grant, in *Indian Annals*)."

* [In this sketch of chronic camp dysentery, as it prevailed in our armies, the writer has availed himself of the statistics and valuable pathological researches of Dr. J. J. WOODWARD, U.S.A., in his Report on the Medical History of the War, Circular No. 6, Surgeon-General's Office, War Department, 1865.]

meat and vegetables were deficient, but also by the presence of well-marked scorbutic phenomena. The influence of climate and season is abundantly proved by the detailed statistics of individual armies and localities. The amount of greatest prevalence in any given army was also the month of the greatest prevalence of intermittent fever.

Like camp fever and intermittents, diarrhoea and dysentery were most frequent in the Central region, less so in the Atlantic, and least in the Pacific region. In the Central region the cases were more numerous than the strength during the first year, and nearly equal to the strength during the second; in the Atlantic they were more than half the strength during the first year, and more than three-quarters during the second; in the Pacific region they were each year somewhat over one-quarter the strength. The differences between the ratio of mortality to strength in the three regions was still more striking. In the Central region there was 9 per 1000 of mean strength during the first year, and 23 per 1000 the second; in the Atlantic 1 per 1000 during the first year, and 9 per 1000 the second; in the Pacific region less than 1 per 1000 during each year. The ratio of deaths to cases shows strikingly the influence of region.

RELATION BETWEEN CASES AND DEATHS OF DIARRHOEA AND DYSENTERY.

	YEAR ENDING JUNE 30, 1862.		YEAR ENDING JUNE 30, 1863.		ANNUAL AVERAGE FOR THE TWO YEARS.	
	Ratio of deaths per 1000 cases.	Number of cases to each death.	Ratio of deaths per 1000 cases.	Number of cases to each death.	Ratio of deaths per 1000 cases.	Number of cases to each death.
Atlantic region, .	2.07	483.09	11.84	84.46	8.60	116.28
Central region, .	9.66	103.52	27.54	36.31	22.85	43.77
Pacific region, . .	2.75	363.64	3.00	333.33	2.89	346.02
Total,	5.55	180.18	20.31	49.24	15.93	62.77

The influence of season in each of the regions was most marked. Dysentery and diarrhoea were by far most frequent in the summer and autumnal months. In the Atlantic region the greatest monthly ratio was during July, 1861, after which it diminished through the fall and winter, but again increased from March to June, 1862; July, 1862, was the maximum month for the second year. The cases greatly diminished in August, after the Army of the Potomac had withdrawn from the Peninsula to near Washington; but increased in September, and attained a second maximum in October, which is the month in which intermittents were most frequent in this region; it then diminished steadily till April, 1863, increasing again in May and June. In the Central region the maximum month is August, 1861, after which there is a gradual diminution till December, an increase in January, 1862, a falling off in February, and a great increase in March and April. The monthly ratio became gradually less during May, June, July, and August, but increased considerably in September, after which it gradually diminished until January, 1863, when it increased again, and made subsequently but slight fluctuations. On the Pacific coast the disease was most frequent during the summer and autumnal months. The following tables give the rates:

MONTHLY RATES OF DIARRHŒA AND DYSENTERY IN THE ARMIES OF THE UNITED STATES DURING THE YEAR ENDING JUNE 30TH, 1862, EXPRESSED IN RATIO PER 1000 OF MEAN STRENGTH.

	1861.						1862.						For the Year.
	July.	August.	September.	October.	November.	December.	January.	February.	March.	April.	May.	June.	
Atlantic region,	88.23	116.29	70.80	62.66	46.06	28.54	23.20	22.20	35.22	67.20	70.92	87.06	646.01
Central region,	93.06	139.99	97.94	94.68	70.91	63.34	71.87	55.53	70.73	107.12	97.99	83.78	1019.54
Pacific region,	28.41	40.06	29.03	27.03	30.85	13.54	10.74	16.54	18.13	16.57	22.93	35.82	271.70

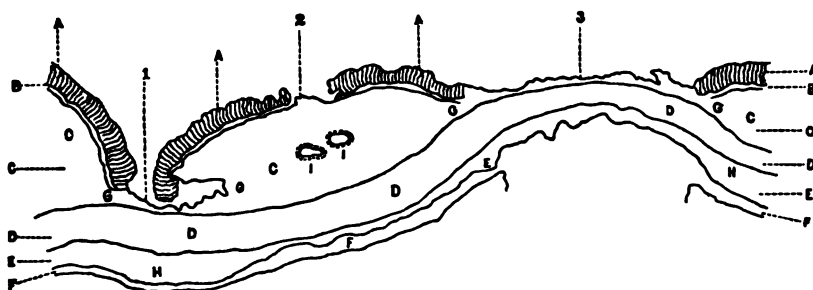
MONTHLY RATES OF DIARRHŒA AND DYSENTERY IN THE ARMIES OF THE UNITED STATES DURING THE YEAR ENDING JUNE 30TH, 1863, EXPRESSED IN RATIO PER 1000 OF MEAN STRENGTH.

	1862.						1863.						For the Year.
	July.	August.	September.	October.	November.	December.	January.	February.	March.	April.	May.	June.	
Atlantic region,	129.56	94.40	105.00	111.22	73.47	63.64	58.50	45.52	38.57	31.85	58.02	59.96	816.22
Central region,	79.76	67.64	88.45	86.93	66.27	60.03	84.54	70.84	76.73	67.86	71.48	86.36	902.62
Pacific region,	38.43	36.21	33.53	28.38	26.97	18.66	14.65	7.55	12.54	18.38	15.09	21.16	267.42

Anatomical Characters.—The pathological anatomy of Chronic Camp Dysentery may be arranged in four groups—(1.) Follicular lesions of the colon, presenting all the transition forms of simple enlargement of the solitary follicles, rupture of the same, and the formation of rounded or oval ulcers, extending nearly or quite to the muscular coat, looking as if they had been cut out with a punch, on a grayish or yellowish-gray base, and sometimes filled with mucus, at other times pus. The enlarged follicles are occasionally the seat of pigment-deposits; and in some cases an areola of pigment, deposited in and among the glands of Lieberkühn, surrounds the enlarged and solitary black follicles; this was found generally in patients who had died of some other disease, as intercurrent camp fever, &c. The colon is usually more or less thickened, even to the amount of a quarter of an inch. Its texture, when cut into, is frequently tough and lardaceous, and often softened. The color of the mucous membrane of the colon is seldom natural, being ash or slate-colored, or greenish-red, reddish-brown, or reddish-black; at times there are patches of congestion. (2.) The extension and agglomeration of the follicular ulcers, by burrowing in the submucous connective tissue, destroy large portions of the mucous membrane by vast erosions. The mucous layer, containing the glands of Lieberkühn, undermined by the extension of the ulcer, not unfrequently hangs in shreds like a fringe from its edge, the undermined portion being occasionally destroyed by ulceration, but more frequently by sloughing. In such cases the mucous membrane is

generally of a slate, dark-red, brownish, or greenish-brown color; the base of the ulcers is yellowish or yellowish-brown, often with brown or blackish sloughs adhering to their surface or edges. This group represents a more advanced stage of the disease. (3.) In addition to the lesions of the first and second groups, the surface of the large intestine is more or less coated with a yellowish, or greenish-yellow, or brownish-yellow croupous, pseudo-plastic, caco-plastic, or false membrane, similar to the membrane formed in the air-passages in diphtheria, and which sometimes extends to the small intestines, and is generally found in those who have died during the supervention of acute dysenteric symptoms. Examination by the microscope of properly prepared sections shows it to be composed of innumerable round cells (lymph-cells, pus-cells), held together by an adhesive granular matrix, more or less re-

FIG. C.

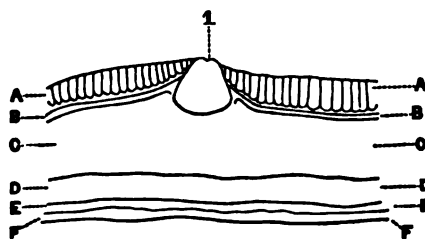


Perpendicular section of ulcerated colon in Chronic Camp Diarrhoea, cut transversely. A A A A. Follicles of Lieberkühn. B B. Muscle of Brücke. C C C C. Submucous connective tissue. D D D D. Circular muscle of the colon. E E E. Longitudinal muscles, cut transversely; at H H, two of the longitudinal bands of this layer. F F F. Peritoneal layer. G G G G. Points of very active cell multiplication. I I. An artery and vein cut through transversely. 1. A deep follicular ulcer, extending to the muscular layer. 2. A small superficial ulcer, which has penetrated to the submucous connective tissue. 3. An ulcer of great size, penetrating nearly to the muscular layer, which is covered with granulations. (Woodward.)

sembling coagulated fibrine. The origin of this membrane may be traced to a rapid multiplication of epithelial cells and superficial connective tissue corpuscles of the diseased mucous membrane.

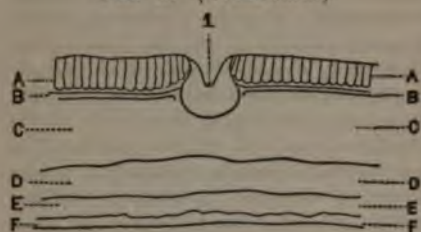
Two forms of ulceration are observed in the colon: in the first, the process begins in the closed follicles; in the second, in the intestinal epithelium or the glandular layer. The closed follicles enlarge by multiplication of their cellular elements till they project as little tumors above the surface, as shown in Fig. D, in which is the enlarged solitary follicle. The tumor, having enlarged to a certain extent, ruptures; its cellular elements escape, and a minute ulcer is formed, as shown in Fig. E, in which is the ruptured follicle, with the minute ulcer. The cells or corpuscles of the connective tissue surrounding the enlarged follicle now multiply, and the ulcer spreads by the superficial cells floating off into the intestinal cavity, while a new base is continually formed by the multipli-

FIG. D. (Woodward.)



cation of the subjacent cells. The second form of ulceration begins by

FIG. E. (Woodward.)



an abrasion or denudation of epithelium at some point which does not correspond to the position of a solitary follicle. The follicles of Lieberkühn are next destroyed, and the ulcer spreads in the connective tissue by the process just described. The central ulcer 1, is of this variety. Such ulcers are rarer than those of the first kind, and probably are always secondary to them.

Metastatic abscesses, resembling those in pyemia after gunshot injuries, are sometimes found, generally in the liver, but also in the lungs and spleen. In seventeen cases Prof. Alonzo Clark found the kidneys more or less diseased; their weight was somewhat increased, and the cortical portion abnormally light and granular. Microscopical examination showed the tubuli uriniferi filled with granules and detached epithelium (*American Medical Times*, Feb., 1863).

Symptoms.—Chronic camp dysentery most generally is preceded by several acute attacks, becoming gradually established. It sometimes follows an attack of fever. When it has fairly set in there are inappetency, sickness, growing debility, mental indifference, progressive and often rapid emaciation, which may become extreme, a dry, harsh condition of the skin, with increased animal temperature, retracted abdomen, with some tenderness, and imperfectly feculent stools, varying from four or six to twenty in the day. The evacuations frequently contain undigested portions of the patient's food; tubular casts of false membrane are to be seen in some cases; and mucus, pus, and blood in variable quantities; with excess, deficiency, or absence of the products of the intestinal transformation of the hepatic secretions. For the recognition of the blood and pus, when present in small quantity, the microscope must be used. It may happen that though pus exists, its corpuscles have been destroyed by the peculiar form of putrefaction which the intestinal contents sometimes undergo. Torula-cells, indicative of the setting up of fermentation in the saccharine and starchy elements of the undigested food, are frequently met with (SALISBURY). Edema, and even serous effusions into the visceral cavities may happen. The urine, often scanty, contains tubular casts, but is usually free from albumen, unless Bright's disease be present. Ulceration and perforation of the cornea have been seen (FLINT). In some cases, generally lengthened ones, acute dysenteric symptoms come on, with fever, delirium, abdominal tenderness, tormina, tenesmus, rapid sinking, and death. Complicating malarious and scorbutic phenomena generally exist. The other complications met with, commonly in fatal cases, are diphtheria, congestive pneumonia, serous apoplexy, Bright's disease, hepatic embolism, and abscesses of the liver. The disease may last for months and even years.

Treatment.—The impotency of drugs in chronic camp dysentery must be admitted by all physicians who have had large experience with it. Dr. Woodward remarks, "The whole range of vegetable and mineral tonics, astringents, and alteratives, have been employed;" and, though sometimes palliative, and controlling, it is very doubtful if they are ever curative. Temporary arrest, or mitigation of the disease, may follow the use of the subnitrate of bismuth, arsenic, nux vomica, strychnia, the mineral acids

(nitric and sulphurous), the nitrate of silver, the oil of *erigeron canadensis*, persulphate and perntrate of iron, &c. When present, as they generally are, the malarious and scorbutic phenomena must be specially treated. Climate is the essential element in the treatment; its influence is absolute and abiding; and without it permanent results are rarely, if ever, attained. The value of climate in chronic camp dysentery was recognized during the Mexican war; and during the late civil war, the Medical Department of the Army was fully alive to the advantages to be derived from it; and it is conclusively shown that the number of recoveries bore a direct relation to the latitude of the climate, and its freedom from paludal poison, in which the patients were treated. Soldiers with chronic diarrhœa were, in the Atlantic region, transferred to the hospitals in the State of Vermont, and in the Central region, to high northern regions, Keokuk, Ia., Madison, Wis., Detroit, &c., and with favorable results.

During the second year of the war, the proportion of deaths from chronic dysentery in the general hospitals of New England, was 1 to every 48.8; New York City and State, and New Jersey, 1 in 18.7; Pennsylvania and Delaware, 1 in 15; Maryland and District of Columbia, 1 in 11.4; the Virginia, North and South Carolina coast, 1 in 7.1. The writer's experience during two years as Medical Director, Department of the South, was that under no medical or dietetic treatment was a single case cured, and but few benefited; the mortality being very great, until the plan of sending all the cases of the disorder, as soon as it became developed, to Northern hospitals. In the Central region the relative influence of latitude was observed, though the mortality was greater, being 1 in 8.53 in the hospitals of Cincinnati, Ohio; and 1 in 4.7 in those of New Orleans, Louisiana. In the hospitals of Keokuk, Ia., the death-rate was 1 in 9.1; in St. Louis, 1 in 5.2; and in Cairo, situated on the alluvial peninsula formed by the junction of the Mississippi and Ohio rivers, and where the most intense malarial influences prevail, it was 1 in 3.98. The diet should be antiscorbutic, and fresh vegetables and ripe fruits are useful; fresh meat, well minced, and broth made from it and not from any *extractum carnis*, with milk, cream, and eggs, should be allowed, according to the state of the digestive organs. The sulphites in weak vegetable bitter infusions, will check the fermentative and putrefactive tendency in the stomach and upper intestines, and aid digestion.]

DIARRHŒA.

LATIN, *Alvus soluta*; FRENCH, *Diarrhée*; GERMAN, *Durchfall*—Syn., *Diarrhœe*; ITALIAN, *Diarrea*.

Definition.—*A frequent discharge of loose or fluid alvine evacuations, without tormina or tenesmus.*

Pathology.—This affection is rather a consequence or a symptom of certain pathological states than of itself a disease; yet, as there are many agents, both of a moral and physical nature, that act thus upon the human body; and as there are also many known morbid poisons which bring about this state, it merits some notice in the class of diseases now under consideration. It is a morbid action of function, rather than any disease of structure, being unassociated with any definite specific lesion of vital parts. It may be regarded

generally as the immediate result of unwholesome diet, excess in food or drink, cold, wet, fatigue, and exposure, and various functional derangements of the biliary and gastro-intestinal apparatus.

Symptoms and Forms of Diarrhœa.—Nosologists have generally divided the disease into varieties founded on the different states of the discharges; but as these do not depend upon definite pathological states, the classification is of little use. Nevertheless, the state of the discharges furnishes important indications in the treatment of the disease. The most common appearances are due to the predominance of fluid feculent matter, or to bile, mucus, serum, chyle, or where undigested masses of food pass unchanged, giving rise to what is termed a "lientery." But the discharges are more often of a mixed kind, made up of several of those states.

The idiopathic forms of diarrhœa which require notice are,—(1.) *Diarrhœa of irritation*; (2.) *Congestion, or inflammatory diarrhœa*; (3.) *Diarrhœa with discharges of unaltered ingesta (lientery)*.

(1.) *Diarrhœa of Irritation.*—This form comprises most of the cases denominated *feculent* by authors. It is induced by stimulating or irritating substances received into the stomach, excesses in eating or drinking, or even by a small quantity of unwholesome food, or by poisoning from animal effluvia and from certain mineral poisons, or what constitutionally disagrees with the patient. In infants it is often brought on by unwholesome conditions of the milk, such as the persistence of *colostrum* in it. Nausea, with severe griping pains before each evacuation, a foul, loaded tongue, copious feculent stools, watery, mucous, or bilious, and becoming frothy, are the phenomena of this form of diarrhœa.

(2.) *Diarrhœa from Increased Vascular Action.*—This variety is caused by whatever induces a greater flow of blood to the intestinal mucous surface, and at the same time lessens or obstructs the cutaneous elimination of fluids; the application of cold to the cutaneous or pulmonary mucous surfaces, or to both at once; cold acid drinks, or ices taken when the body is overheated; suppression of perspiration or of accustomed discharges; checked menstruation or lochial discharge.

The evacuations are watery or serous, with mixed feculent matter, and exhibit every shade, from a dark brown, greenish-brown, to a pale grayish or whitish color; and they contain, in some cases, pieces of thick gelatinous mucus, or thin, glairy, and stringy mucus. In other instances, whitish, albuminous flocculi are abundant in the stools; and in a few instances large membranous or albuminous shreds or flakes present a mould of the internal surface of the gut.

In addition to the symptoms noticed in the former variety, there is, in this form of diarrhœa, a dry, harsh skin, with increased temperature of the trunk, a flatulent state of the bowels, a small, frequent, constricted, but soft pulse, a furred or loaded tongue towards its root, with red edges and point, and scanty, high-colored urine. In infants this variety is known as the "watery gripes," and often precedes fatal exhaustion in them.

(3.) *Diarrhœa with Discharges of Unaltered Ingesta.*—This is essentially an atonic form of diarrhœa, and very different from the last

variety. It corresponds to the "*diarrhœa lienterica*" of the older authors. The most marked and characteristic phenomena which attend the disease are due to the almost total suspension of the digestive, assimilative, and absorbent functions, the *egesta* often differing but little in appearance from the *ingesta*. Such a form of diarrhœa occurs more frequently in children before the period of the second dentition than at later periods. It is frequently the consequence of previous inflammatory irritation of the alimentary mucous surface, and disease of the mesenteric glands. It seems as if, in this variety, the stomach had lost its tone or vital energy, as well as the mucous membrane of the alimentary canal; and it no doubt results, in the first instance, from indigestion. This was a frequent form of diarrhœa amongst the soldiers in the Crimea, as observed by Dr. Lyons; and the soldiers themselves observed it, and were in the habit of saying, "It is of no use eating, as our food passes through us in the same state as it goes in." The appetite is usually voracious; and when this form of diarrhœa continues long, the debility becomes extreme; and when death takes place, it is from stupor and exhaustion.

In a practical point of view, these are the principal varieties of idiopathic diarrhœa which require to be distinguished; and the diagnosis of the form of diarrhœa symptomatic of the invasion of other diseases are noticed under the special diseases of which they form a part.

Treatment.—For practical purposes, the treatment of these three forms of idiopathic diarrhœa may be founded on the following indications—namely, *first*, that in which the *tongue is clean*, the pulse quiet, and all constitutional reaction absent; and, *second*, that in which the *tongue is white* and coated, the pulse accelerated, some fever present, and the pain or soreness constant and increased by pressure. The stools in either case may be black, green, white, or mixed with blood indifferently.

When the *tongue is clean*, if the disease be quite incipient, the usual practice is to give one dose, consisting of an opiate, combined with a gentle cathartic. The form may be *one grain of opium*, combined with a *drachm of compound rhubarb powder*, or combined with *five grains of calomel*. To remove any offending matter that may be present, their action may be aided by castor oil, or a saline cathartic, such as a Seidlitz powder. Sometimes it may be advisable to omit the opium, and to combine antacid remedies with the purgative, as in the following prescriptions:

R. Sodæ Bicarbonatis, Hydrargyri cum cretâ, ãã gr. ij—ad gr. v;
Magnesiæ Carbonatis, gr. iij—ad gr. vj; Pulv. Rheï, gr. v—ad gr. viij;
misce.

Or:

R. Sodæ Bicarbonatis; Pulv. Rheï; Pulv. Calumbæ, ãã gr. iv—ad gr. vi; *misce.*

The administration of such a powder may be repeated at intervals—twice or thrice a day; and ipecacuanha in small doses (a

quarter or a sixth of a grain) may be sometimes advantageously combined with each dose. These medicines having produced their intended effect, others more distinctly astringent may be administered if the diarrhœa persists. In many cases a drachm of *syrup of poppies* after each stool is sufficient. In severe forms of the disease a scruple to half a drachm of the *compound chalk powder*, in some aromatic, such as peppermint or cinnamon-water, every four or six hours, is an excellent remedy; and these medicines may be used whether blood be or be not in the stools. If the opiate and aromatics contained in the above medicine should prove insufficient, it may be necessary to add to each dose some of the class of pure astringents, as a drachm of the *tincture of kino* or of *catechu*, or *hæmatoxylin*, or of *iron*.

There are cases of diarrhœa with a *clean tongue*, which will not yield to opiates, astringents, or stimulants, either singly or combined, and which probably depend on a want of tone in the intestine; and in these cases *five grains of salicine* every four or six hours have often stopped a diarrhœa that appeared fast hurrying the patient to his grave. *Tincture of sesquichloride of iron* is similarly useful, in doses of five to ten minims.

When diarrhœa is accompanied by a *white furred tongue*, together with pain and soreness, it is necessary to give opiates, combined with some mild purgative. Thus, *half a drachm to a drachm of Epsom salts* with a *drachm of the syrup of poppies*; or *fifteen minims of the tincture of hyoscyamus*; or, in severe cases, with *three to five minims of tincture of opium*, every four or six hours, are remedies on which, as a general principle, we may very confidently rely. In other cases, *rhubarb*, *castor oil*, or any other *mild purgative*, may be substituted for the Epsom salts. In cases of diarrhœa accompanied by vomiting, a *drachm of syrup of poppies alone*, repeated every half hour, or every hour, for two or three times, may quiet the stomach, and enable it bear the other remedies; or soda-water, or the effervescing draught, with a tablespoonful of brandy, with or without a few minims of tincture of opium, often remain on the stomach when everything else is rejected.

Most practitioners lay great stress on the color of the stools, and the necessity of correcting the supposed morbid states of the liver; but the various colors of the stools are in many instances caused rather by morbid secretions from the surface of the mucous membrane of the intestines than by any defective state of the bile in the gall-bladder; and the conclusion from this consideration is, that, in simple diarrhœa, mercury (which is so often given in a routine way) in any form is either unnecessary or injurious in the majority of cases, except as a purgative. In a smaller number, however, it is sometimes necessary, and more especially in children under four years of age. One general rule may be acted on in the cure of diarrhœa, which is, that in the adult, whatever be the form of the diarrhœa, if the stools be dark at first, and then become light-colored, purgative medicines are no longer beneficial, and in no instance ought they to be continued longer than is sufficient to remove any irritative substance accumulated in the alimentary canal.

Sulphuric acid, in doses of the officinal diluted drug, of twenty to thirty drops, with water simply, or combined with the compound tincture of gentian, has been found a useful remedy. The sulphuric acid may be alternated with the nitro-muriatic acid, and prescribed in a similar manner.

The dietetic treatment should be limited to slops, puddings, and whitefish boiled, and the drink to weak brandy and water, which acts locally as an astringent, and generally as a diffusible stimulus.

MALIGNANT CHOLERA.

LATIN. *Cholera pestifera*—Idem valent, *Cholera serosa*, *Cholera spastica*, *Cholera asiatica*; **FRENCH.** *Choléra asiatique*; **GERMAN.** *Cholera nostras*—Syn., *Cholera*, *Asiatische Cholera*; **ITALIAN.** *Colera morbus*.

Definition.—A disease essentially of miasmatic origin, developed under certain atmospheric and terrestrial local conditions in Europe, Asia, and America, and capable of being propagated or diffused, to a certain extent, over the surface of the earth, through the atmosphere, or in some other way, and also by means of human intercourse between the healthy and the sick. It is characterized in many (but not in all) cases by premonitory diarrhœa, sudden muscular debility, tremors, vertigo, occasional nausea, and spasmodic pains in the bowels, depression of the functions of respiration and circulation, and a sense of faintness; copious purging of serous fluid and sometimes blood, succeeded by vomiting and burning heat at the stomach, coldness and dampness of the whole surface of the body, coldness and lividness of the lips and tongue, cold breath, a craving thirst, a feeble rapid pulse, difficult and oppressed respiration, with extreme restlessness (a state expressed in physiological language by the term “*anxietas*”), suppressed urinary secretion, blueness of the entire surface of the body, a sunken and appalling countenance, a peculiarly suppressed voice, a peculiar odor from the body, partial heats of the præcordia and forehead—fatal collapse, or reaction and secondary fever. Under conditions favorable to its development it often becomes epidemic.

Pathology.—During recent years volumes have been written on the nature and causes of cholera; and the description of it which I here give is chiefly drawn from the writings of men who, while they have been themselves original observers of the disease throughout an extensive experience in this country, in India, and in the north of Europe, have since been the able expositors and philosophical critics of the numerous official, original, and independent scientific accounts of it which have been lately given to the world.

The remote cause of cholera is unquestionably a poison; and that it should spread over countries which, in respect to climate, soil, geological formation, and also as to the moral and physical habits of the population, are the most opposite to those where it first originated, is only explicable by the hypothesis of its propagation by a specific poison.

One doctrine, therefore, now very generally accepted regarding

the pathology of cholera, is that a poison *has been absorbed*, and infects the blood; that, after a longer or shorter time, it produces a primary disease of the blood; that it undergoes enormous multiplication in the living body of the cholera patient, as a result of the morbid process so established; and that changes are induced in the function of respiration directly consequent upon this alteration of the blood by the poison.

" Whose effect
Holds such an enmity with blood of man,
That, swift as quicksilver, it courses through
The natural gates and alleys of the body;
And, with a sudden vigor, it doth posset
And curd, like eager droppings into milk,
The thin and wholesome blood."

HAMLET, Act I, Scene V, line 64.

So wrote our greatest English poet three hundred years ago; and by such characters it has been ably shown by Dr. Wm. Budd, of Bristol, Sir Thomas Watson, and others, that cholera identifies itself with that group of contagious poisons which gives rise to acute general diseases. It is this multiplication, and the disturbance to the vital functions, which in each case constitutes the disease and destroys life. In small-pox the work of reproduction is seen in results directly appreciable to the eye. An impalpable speck of small-pox virus inserted into the skin may produce a disease which, in the course of a few days, issues in the development of a new stock of the same virus, sufficient in amount to inoculate myriads of other persons with small-pox; and although the fact may not yet be open to evidence so precise, yet the circumstantial evidence is conclusive which shows that, in any case of Asiatic cholera, its specific poison is multiplied in a ratio at least as great.

But another doctrine is now also making progress—namely, that the blood is only *secondarily affected*, and that *primarily a poison* (of fungoid? nature) makes its way to the intestinal mucous surface, and there develops itself, to the rapid annihilation of the powers of life. If such a doctrine proves true, it will cause this disease to be classified amongst parasitic poisons.

A peculiar character regarding the cholera poison which is very necessary to be remembered when we try to interpret the varied anomalies associated with local outbreaks, is the *tendency of the poison, when in a moist state, to rapid spontaneous decomposition and extinction* (Budd, *Associat. Journal*, 1854). This characteristic property accounts in some measure for the rapid subsidence and short duration of particular epidemics; and it does not seem that the poison can long subsist in the common conditions of the English climate. In India, on the contrary, where the disease may be said to have had its birth, the atmospheric conditions appear to be more favorable to the preservation of the powers of the poison—partly from the nature of the temperature, partly from the climate being such as rapidly to dry up the poison-holding material—so that its essence is unimpaired.

The phenomena resulting from the changes in the blood in cholera are the proper and distinctive symptoms of the disease; and the

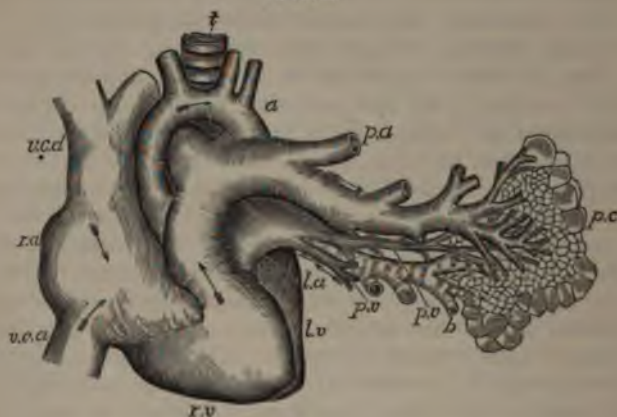
term "*algide*," first used by the French pathologists, very properly designates one of its most remarkable and constant symptoms—namely, the diminution of animal heat. The sensation of cold communicated to the observer has been compared to that experienced on touching a moist bladder or the skin of a frog. The *algide* symptoms, therefore, essentially constitute the characteristic phenomena of this disease. In proportion to them is the malignity and rapidity of the case. They afford the only measure of its severity, and from them only can a correct prognosis be formed. The vomiting, purging, and cramps are considered as non-essential phenomena; for authentic cases of cholera are on record entirely divested of these symptoms; and the suddenness with which the poison sometimes extinguishes life is extremely remarkable. Instances of death taking place in two, three, four, or six hours, are by no means uncommon. When the disease broke out at Teheran, in May, 1846, Dr. Milroy states that those who were attacked dropped suddenly down in a state of lethargy, and at the end of two or three hours expired, without any convulsions or vomitings, but from a complete stagnation of the blood. In Bulgaria, during the outbreak of cholera in the allied armies, in the summer of 1854, the rapidly fatal character of the early cases was notorious. Such records confirm the views developed by Drs. Parkes, Johnson, Sir Thomas Watson, and others, as to the essentially poisonous nature of the disease, and the very rapid depressing influence of the poison. It is plain, also, that a poison so powerful, so suddenly overwhelming all Nature's efforts at resistance, does not allow time, in many cases, for any secondary or specific actions to be set up.

After death, during collapse, the following lesions have been noticed: The follicular structure of the intestinal canal has been found to be swollen, and the intestine filled more or less with a turbid, inodorous, semi-diaphanous fluid, usually compared to a thin starch or rice-water, the remains of that immense secretion which has taken place during life, and which, being tested, has been found sometimes acid and sometimes alkaline. It is found in its most unmixed condition in the small intestines. It consists of a thicker and thinner portion, and it appears to be the latter which chiefly constitutes the "rice-water" stools, which may be passed off without admixture of the thicker substance. A layer of grayish mucus has also been found coating the whole of the mucous membrane of the alimentary canal, but without a trace of bile, although the gall-bladder is usually filled with that fluid. If the first stage has been prolonged, the mucous membrane of the alimentary canal is of a livid color, and in some instances has presented a mammillated appearance, caused by an enlargement of the tubular glands, from which a white opaque fluid can be squeezed out and the mammillated appearance effaced.

The liver, the spleen and the kidneys have in general been found gorged with blood as to their veins, and the veins of the kidneys are quite as visible as from "contracted mitral-valve" disease (Sutton); and this engorgement extends even to the bones, which, Louis says, appear as if the animal had been fed on madder. The capillary

vessels were empty. Professor W. T. Gairdner considers this state the natural appearance in persons dying of such very acute diseases as do not remove the coloring matter from the blood (*Edin. Med. Journal*, July, 1849). The bladder is contracted and empty. The membranes of the brain and cord are in general congested, and the substance of the brain dotted with more *puncta cruenta* than usual. The most common appearances in the lungs are the presence of blood in the ramifications of the pulmonary arteries, chiefly or solely; the collapse and the deficient crepitation—arising from the more or less complete absence of air in the air-cells and blood—in the minuter capillaries, and from the approximation of the molecular parts of the pulmonary substance. The ultimate tissues of the lungs are pale and bloodless. In other cases there is more blood in the minute structure, a corresponding dark color of the lung, and a variable amount of frothy serum. The right side of the heart and the pulmonary arteries were generally filled, and, in some cases, distended with blood; the left side and aorta were generally empty, or contained only a very small quantity of dark blood; the left side evidently had received little or no blood; but had continued to contract, in some cases even violently, on the last drop of blood which had entered it (so-called “concentrical hypertrophy”). It was curious, also, to notice that the icy coldness of the body in the stage of collapse passed away after death, when the temperature is said to have risen sometimes to 102° or 104° Fahr. Dr. Johnson gives

FIG. 1.*



the annexed diagrammatic representation of the state of the heart, lungs, and large vessels, when death has occurred during the stage

* EXPLANATION OF THE FIGURE (after Dr. Johnson).—The venæ cavae, the right cavities of the heart, and the pulmonary artery with its branches, are seen to be distended; while the pulmonary capillaries, the pulmonary veins, the left cavities of the heart, and the aorta with its branches, are comparatively empty: (v. c. d.) vena cava descendens; (v. c. a.) vena cava ascendens; (r. a.) right auricle; (r. v.) right ventricle; (p. a.) pulmonary artery; (p. c.) pulmonary capillaries; (p. v. p. v.) pulmonary veins; (l. a.) left auricle; (l. v.) left ventricle; (a.) aorta; (t.) trachea; (b.) bronchus.

of collapse in cholera, from an original drawing by Mr. John Wood. The lungs weighed very much less than usual, often not half their usual weight; and on section they appear dry. The blood they contain is all in the branches of the pulmonary arteries: it is black-looking blood—thicker than usual, but still fluid, so that on puncturing a vein, such as the jugular, it escapes in such quantities that the right side of the heart is emptied in a few minutes (SUTTON). The anterior portions of the lungs were of a gray color—very much paler than normal: the posterior portions and the bases of the lungs were much darker in color, soft in consistence, and easily broken down. A section of the lung substance rapidly became of a bright scarlet color on exposure to the air.

Such are the appearances which the body has presented when the patient has died in the first, the asphyxiated, pulseless, or collapse stage. The enlargement of the solitary follicles of the intestines is believed to be peculiar to those cases in which diarrhoea, or other disorder of the alimentary canal, had for some time preceded the fatal attack. This enlargement bears no relation to the intensity of the disease, being often most conspicuous in the least severe cases; and it is an appearance now considered of secondary importance, and consequent on the purging. In the experience of Dr. W. T. Gairdner it has been found in about two-thirds of the cases.

When the patient has survived until reaction has taken place, and the second or febrile stage has been formed, the body no longer presents that shrunk, worn, and livid appearance it did on death taking place in the first stage; but, on the contrary, rather a full and plump appearance. The injection of all the large organs disappears, the blood being recalled to the surface of the body. The alimentary canal is no longer distended with the turbid secretion peculiar to cholera, but contains a thin yellowish *purée* of fecal matter, having the usual odor. The mucous membrane of the alimentary canal has now, however, been found more or less diffusely inflamed, sometimes in all its divisions, but more especially in the pyloric portion of the stomach, and also in the duodenum. The glands of Peyer, as well as the solitary glands, though occasionally found enlarged, were seldom found ulcerated; but when that was the case the corresponding mesenteric glands were also enlarged, being sometimes pale or purple, and when cut into, gave issue to a dark liquid blood.

The post-mortem appearances, and the order of the symptoms tend to show that the blood is obstructed in its passage through the lungs; and that the loss of animal heat, embarrassment of the respiration, and gradual arrest of circulation, are produced by some aberration of the proper respiratory changes, or impediment to them. But as the mechanical part of respiration remains perfect, and as there is no impairment in the voluntary command of the respiratory muscles, and as the heart evidently beats in many cases till stopped by the want of blood on the left side, and by its accumulation on the right side, "we are compelled to look," says Dr. Parkes, "for the cause of such arrest of the circulation in the only remaining element of respiration—namely, in the blood itself."

(*Researches into the Pathology and Treatment of Asiatic or Algid Cholera*, p. 107). Dr. Parkes and Dr. Garrod, of London, and Schmidt, of Dorpat, have made the most important researches on the chemistry of the blood in cholera. The latter observer has attempted to trace out the exact chemical steps which attend the period of transudation from the blood into the intestinal canal. The most prominent phenomena of cholera, during this period of transudation, consists in "separation of the water and of the salts of the intercellular fluid (of the blood) through the mucous membrane of the intestinal canal, and the retention in the blood of an important excess of albumen, and of blood-cells, with apparently less, but in reality with great diminution of the salts and fibrine." The period during which this transudation takes place is generally one of definite duration (about thirty-six hours), and in it the serum and fibrine (intercellular fluid of the blood) are first affected. Water, salts, and a small portion of albumen, pass off, and form the well-known liquid stools. The order in which the constituents of the serum are affected is thus stated by Schmidt: The water transudes before the solids of the serum; the inorganic before the organic solids; the chlorides before the phosphates; the salts of soda before the salts of potash; and the order is very much the same as takes place during the action of purgative medicines, such as *elaterium*. Very soon after this transudation commences, an important change occurs in the blood: the normal diffusion currents between its fluid part and the fluid in the blood-cells alter; and the constituents of the blood-cells transude into the serum, in the same order as the constituents of the serum transude into the alimentary canal; that is to say, the water diffuses more readily than the solids; the inorganic solids more readily than the organic; the chlorides (and of these the soda salts) more readily than the phosphates. The result of all these changes in the fluid of the blood, and in the blood-cells, is, that at the height of the transudation-period the constitution of the blood is profoundly altered. The inorganic constituents, if compared to the water, are during the first four hours increased, because at this time the water is passing off with great rapidity; afterwards, as the salts pass off, the disproportion is lessened, and after eighteen hours or so, the proportion of salts is greatly diminished, and, if compared with the organic constituents, the diminution is enormous. With respect to the individual salts there is in the blood a relative preponderance of phosphates over chlorides, and of potash salts over soda salts. By the end of eighteen hours or so, the blood-corpuscles are left in a most abnormal condition. The great loss of water and of salts, especially of the chloride of potassium (a most important constituent of the blood-cells), at once leads to the conclusion that their functions must have been greatly impaired. Schmidt accordingly found that the amount of oxygen contained in them was lessened by one-half.

According to the observations and analyses made by Dr. William Robertson, of Edinburgh, the fibrine of the blood is usually in large amount, and coagulable with great firmness. On the other hand, defective or imperfect coagulation of the blood in cholera was ob-

served by Dr. Parkes as occurring in little less than a quarter of the whole number of cases observed by him. The presence of fibrine in the blood was not indicated by any coagulation either in or out of the body; and, whether coagulated or not, the blood has usually a dark color; but it generally acquired an arterial tint when brought into contact with the air in thin layers. Dr. Robertson's observations were made on the cases occurring in the Edinburgh epidemic of 1848 and 1849; while Dr. Parkes's observations were made on two severe epidemics of cholera in India in 1843 and 1845. He also made the interesting observation, that a few drops of the thick substance taken from the intestines had sometimes the effect of restoring the vivid arterial color of the blood. During the transudation into the intestinal canal, it appears that the diffusion currents *from* the blood into various structures are diminished; while, on account of the density of the blood, the inverse currents *from these structures to* the blood are augmented in rapidity. In this way fluids are drawn from the muscles, the viscera, and, in fact, most of the tissues; and it is probable that these fluids are charged with substances (such as sugar, &c.) which, under ordinary conditions, are taken very much more slowly into the blood, and are soon decomposed when they get there. The extent to which the blood is contaminated and injured by this admixture, and also by the retention of urinary constituents, is not yet accurately known. "When we remember," says Dr. Parkes, "the great share taken by the blood-globules in the respiratory and heat-furnishing processes, it is scarcely possible to avoid concluding that their loss of salts is connected with the characteristic cyanosis and lower temperature in cholera. In *most* cases there is vomiting and purging before there is loss of heat, though this very soon follows in a slight degree, and then gradually augments. In other words, the diarrhoea coincides with the first or early chemical changes in the blood—the transudation of some of the constituents of the serum. The lowered temperature follows afterwards, at the time when we know that diffusion from the blood-cells into the serum must be taking place, and augments gradually as the diffusion increases." In all the cases examined by Dr. Marcus at Moscow, in 1832, the clot and serum evinced acid qualities on the application of litmus, except in four cases, where the discharges were watery and the reaction alkaline. The phenomena of the disease may thus be traced from the transudation of serum constituents as the starting-point. All the other chemical changes in the blood, and the most marked symptoms (such as the abnormal respiratory process), follow as a matter of course.

But the question may be also put in another form, as it has been already so well put by the able reviewer of Dr. Johnson's book in the *Saturday Review*,—"Why has the circulating blood stopped here (in the pulmonary arteries), and by what means has it been brought to a stand? Were the arrest of motion due to gradual thickening in consequence of the continued abstraction of its liquid portion, it would be found stagnating in the capillaries, as well as in the arteries. It must be borne in mind that one characteristic symptom of cholera—that symptom which, irrespectively of the

fatality of the disease, renders it truly a disease to be dreaded—consists in very painful cramps of the larger muscles of the body. These contractions, it may be assumed, are produced by the choleraic poison, just as we know they are producible by the poison of strychnine. Dr. Johnson supposes that a similar spasm or cramped state of the muscular fibres which embrace, and by their natural contractions regulate the size of, the minute pulmonary arteries, is caused by the same choleraic poison, and bars these slender channels against the advancing blood. The thickening of the blood is a consequence, and not a cause, of the collapse. Precisely in the same way does a similar condition of the muscular fibres of the smaller air-tubes of the lungs constitute a fit of spasmodic asthma. . . . Thus the emptiness of the systemic arteries accounts for the extinction of the pulse at the wrist, for the cadaverous sinking in of the eyeballs and falling of the features, for the blueness and coldness of the skin, and for the absence of syncope. The circulation stops, not from debility of the heart, as in exhaustion, but in consequence of a direct mechanical impediment to the onward course of the blood. We can understand the impotence of brandy against this condition; and how, on the other hand, bleeding may help, both by relaxing the spasm and by unloading the distended right heart, to restore the circulation. Into this explanation Dr. Johnson presses, plausibly enough, the singular effect of the injection of fluids into the veins of these patients. It appears that, to be influential at all, the fluids must be hot; and he concluded that they act chiefly by relaxing, through their warmth, the spasm of the smaller arteries. The blood then flows on again, and the symptoms of collapse are for a time removed. Again, the husky whispering voice is owing, not to muscular weakness, but to the small volume of tidal air in the respiratory current. As but little venous blood reaches the lung-tissue proper, there is but little demand for air to meet and decarbonize it. The respiration accordingly becomes shallow, and the vocal pipe, feebly blown through, refuses to speak. Under the temporary impulse of the warm injections, the voice regains its usual tone and note. Once more, there are chemical and less obvious changes already noticed which receive their explanation from this theory."

Such is the nature of cholera, according to the observation of Drs. Parkes, William Robertson, Schmidt, Johnson, and Sir Thomas Watson; and thus "an early theory of the nature of this disease has received the support of the best physicians and chemists of the day—namely, that the blood, if not the primary seat of the disease, becomes eventually contaminated by the action of a specific poison."*

* "The belief," writes Mr. Simon in his *Report on Public Health* for 1866, "that the proximate cause of cholera is a 'poison' first acting in the blood is common to Dr. Parkes and Dr. Johnson, as well as to many other writers; and Dr. Johnson builds on that belief his advocacy of a particular principle of treating cholera—the principle, namely, of 'assisting nature,' by emetics and purgatives, in what he deems to be her 'salutary and curative efforts' of vomiting and purging; but Dr. Parkes's doctrine of the state of the circulatory system in collapse, and Dr. Johnson's doctrine of the dependence of that state on spasmodic closure of the minute pulmonary

Origin and Modes of Propagation of Cholera.—One of the most interesting facts brought out by Dr. Farr and Mr. Radcliffe is, that

arteries, are doctrines which do not necessarily involve an acceptance of the 'eliminative treatment' of cholera, nor presuppose any belief that cholera begins as a blood-disease. It is important that the different questions should not be jumbled together as one; particularly important now, because the notion of a primary blood-poison in cholera seems tending more and more to be superseded.

"1. First, as regards the state of the circulation in collapse. Personally knowing Dr. Parkes's great accuracy of statement, I attach (says Mr. Simon) the utmost importance to the descriptions contained in his work. And their details do certainly in great part justify the generalization which he makes of them.* But whether the morbid phenomena which he describes are rightly accounted for by the doctrine of arterial obstruction in the lungs (either such as he supposes, or such as Dr. Johnson supposes) is matter of much more doubt: for feebleness of heart-contraction appears to be an invariable fact in choleraic asphyxia; and so far as this affects (or at least predominantly affects) the right side of the heart, so far it tends to produce much such a disturbance of circulation as would result from the supposed arterial obstruction. Present opinion seems, I think, generally to be that, in the main, it is the dynamical affection of the heart (not the supposed obstruction of pulmonary arteries) which gives the true explanation of Dr. Parkes's facts; but this would not of necessity imply, either for the pulmonary or for the aortic circulation, that all the arterial resistances are normal. Whatever etiological view be taken of the connection of the symptoms of collapse, it cannot be deemed unlikely that a much diminished volume and impaired fluency of the blood, when they have arisen, should excite certain phenomena of their own in the sphere of arterial contractility, as well as have their own physical consequences; nor, again, that certain changes of arterial tone should go with certain changes of cardiac action. Be this as it may, some of the phenomena presented in the aortic system in collapse are such as arterial contractility would seem very plausibly to explain. Such are some of the inequalities of temperature and circulation in the diseased body, not only as between internal and external parts, but as between different parts (external or internal) in the aortic circulation. Specially, for instance, I cannot conceive from what other basis to explain the tendency to equalization of temperature between external and internal parts which is apt to show itself in the fatal ending of collapse, and even to continue after death, as though a final relaxation of arterial rigidity permitted the blood at last to find way through its normal channels. And if the cold and cyanosed state of external parts in cholera be not to some extent under control of arterial contractility, I cannot conceive through what mechanism to explain that exceptional state of mammary circulation which permits the continued secretion of milk by nursing women who are in collapse.

"2. The belief that a primary 'blood-poison' is the proximate cause of cholera, the direct source equally of its intestinal and of its asphyxial manifestations, is, so far as I know, mere hypothesis. It has been much accepted as the only possible explanation of certain supposed, but very questionable facts in the natural history of the disease; specially in explanation of the supposed fact, that the utmost collapse of cholera may concur with little or no affection of the intestinal canal. It is of supreme pathological importance to be right in the matter of these premises. Is it, or is it not, true that choleraic asphyxia can arise otherwise than in consequence of the bowel disease? This question has been much perplexed, partly through the vast number of vague assertions which are current on the subject, and partly through an assumption which has often been prematurely, and perhaps wrongly, made, that the significance of the bowel disease in cholera is to be measured by the quantity of the fluid secretion from

* "Yet it deserves notice," says Mr. Simon, "that even among Dr. Parkes's own cases of death in collapse the post-mortem evidence of interrupted pulmonary circulation was not universal; and I may add, though without attaching equal importance to the fact, that a citation of miscellaneous authorities on the state of the heart and lungs in death by collapse (such a citation as was given in 1883 by Prof. Phœbus in chapters v and vi of his classical *Leichenbefund bei der Orientalischen Cholera*) would show still less uniformity of evidence in that respect. Also, in my opinion, the assertion made by Prof. Griesinger (in § 483 of the admirable essay on cholera which forms part of his *Infections-Krankheiten in the Handbuch der Pathologie und Therapie*) deserves much weight, viz., 'that the distension of the right cavities of the heart appears not to be present during life, as percussion gives (invariably?) a small area of cardiac dulness.' Supposing the general accuracy of Dr. Parkes's descriptions to be conceded, judgment must, I think, be reserved on the meaning of the alleged exceptions. For, whatever question there may be as to the inter-dependence of the symptoms of cholera, it is certain that the disease, in proportion to its flux, tends to reduce more or less rapidly both the volume and the fluency of the blood; and till we know exactly what would be the ultimate anatomical expression of those changed physical states of the blood, taken by themselves, it is impossible to affix a right value to the cases of cholera where post mortem appearances, or facts observed during life, have not answered to Dr. Parkes's general description of the anatomy of death in collapse."

since the first great outbreak of epidemic cholera in this country in 1832-33, an enormous development of diarrhœa, summer chol-

the bowel. Properly to discuss the main question, that assumption must be disallowed, and the points be separately considered.

"a. That the large, often enormous, fluid discharges which generally characterize cholera represent corresponding *de-aquation* and *de-salination* of blood and textures in the patient's body, and that such changes must at least for a time interfere to some considerable extent with all or most of the chemical processes of the body, cannot, I suppose, be disputed. And on the hypothesis that cholera begins in the bowels, it might seem probable that all, or nearly all, the facts of collapse and secondary fever would admit of being referred, directly or indirectly, to that generally enormous flux. Especially it would seem plausible to refer to the altered blood either a power of mechanical obstruction, or a power of provoking resistant muscular constriction, in the vessels through which it has to pass. At present, however, very strong arguments against any such doctrine of collapse are adduced. Dr. Goldbaum's conclusion, in his recent report (published in *Virch. Arch.*, Feb., 1867) of the experiences of the Berlin Cholera Hospital No. III, in the epidemic of 1866, supported by many illustrative cases, is strongly against the doctrine that inspissation of the blood is the cause of the asphyctic state. He (like many previous observers—notably the chief Anglo-Indian authorities, and in Europe, Magendie, Romberg, Parkes, and others) insists that the relation of flux to asphyxia is rather an inverse than a direct proportion, and that the cases of worst augury are cases which have fallen into collapse after but little or no vomiting and purging. He, moreover, expressly guards against undue importance being attached in such cases to the quantities of fluid (half or two-thirds of a gallon at the utmost) which may be retained within the patient's intestinal canal; pointing out that the contrast is with cases where perhaps as much as seven gallons are discharged by vomiting and purging, and that consequently no allowance made for intestinal contents can affect the truth of his proposition. In this context, too, it seems convenient to refer to the comparison which many experienced observers of both sorts of disease have drawn between the phenomena of cholera collapse on the one hand and those of the cold stage of malarious disease on the other. Dr. Goodeve, for instance, in his article on cholera in *Reynold's System of Medicine*, arguing that 'symptoms similar to collapse may be produced by poisons without any purging,' observes that he has 'seen people under the influence of malarious poison in Calcutta lie for hours as cold and pulseless, and as embarrassed in the breathing, as in cholera.' No doubt both sorts of collapse have very much in common as regards their spheres of manifestation, and much also as regards the phenomena themselves; but of course the likeness between them does not exclude the possibility that they may be induced by very different causes.

"b. As regards the other point, materials for judgment are less definite; but certainly, in the present state of information on the subject, the proposition is by no means established that cholera collapse ever occurs without bowel disease enough probably to account for it. Abstraction of fluid, I need hardly observe, is not the only way by which abdominal lesions can affect the circulation of the blood. There are channels for nervous as well as for humoral sympathy; and the heart's action can be lowered to the utmost (whether with consensual changes of arterial tone, I know not) by abdominal lesions in which little or no fluid is expended. Physiologists will remember those admirable researches of Goltz* and Bernstein,† which elucidate the exact course and mechanism of such sympathies; and every practitioner of medicine or surgery can recall instances where he has seen mortal collapse (substantially, so far as I know, not different from the collapse of cholera) produced by the very onset of traumatic and other abdominal inflammations, sometimes of no great apparent magnitude. In comparison with some of such instances, the least amount of bowel disease which (so far as I know) has ever yet been found in the bodies of persons dead with the cholera collapse, must, I believe, be deemed very considerable. Doubtless there are cases on record where men stricken with cholera collapse are said to have suddenly fallen, even numbers of them together, in the streets or elsewhere in their ordinary pursuits, 'tumbling over each other lifeless,' or as if 'knocked down dead by lightning,' or 'as if they had drunk the concentrated poison of the upas tree.' It may well be that some of these pictures are unintentionally overdrawn; representing less the real objective occurrences than they represent that utter dismay, that sense of

* *Virch. Arch.*, vol. xxi.

† *Reichert and Du Bois Reymond's Archiv.*, 1864.

era, and diseases of a choleraic character, have been unusually fatal in the metropolis and England generally; that the mortality from

mysterious death too swift for remedy, which severe epidemics of cholera are singularly apt to produce. But, taking them at what they are worth, what reason is there to believe that the sufferers who were so stricken down had not bowel seizure as the ground of their collapse? No doubt the opinion has been current that cholera, acting in some mysterious way on the total organism, may 'kill and leave no sign;' but in proportion as exact morbid anatomy has been cultivated, that opinion has, I think, more and more seemed to rest on a mythical basis; and the doctrine of primary collapse ought at least, without hesitation, to be rejected for cases where post-mortem examination of the bowels has not been made. In illustration of these remarks, I would refer very particularly to the important case given by Dr. Sutton, in his report *On the Clinical Characters of Cholera*, 1866. It was a typical case of *cholera sicca*. It was a case of cholera death so swift that probably none of the reported 'upas poisonings' were swifter. But fortunately the body was anatomized. The whole length of the small intestine was found containing choleraic effusion; and to assume, in the face of that fact, that the cholera collapse was primary, would, in the present state of knowledge, be, to say the least of it, a simple *petitio principii*.

"In the present state of knowledge, then, I do not find it proven, nor do I see any theoretical convenience in taking for granted, that cholera begins as an active blood-change capable of producing primary collapse. The facts, so far as I know them, can all be reconciled with the belief that cholera begins as bowel disease, producible by direct contagion, without even a passive intervention of the blood, and that all asphyctic phenomena of the disease are supervenient sympathetic phenomena. That, so far as they are facts of cardiac paralysis and arterial contraction, they may be attributed to nervous sympathy between bowels and circulatory system, without reference to the greater or less humoral effects of the coincident flux from the bowels, is at present a tenable view. At the same time, I hesitate to accept as proven that cholera collapse is independent of humoral sympathy. That it may often be apparently so is, no doubt, well shown by the statements I have quoted from Dr. Goldbaum and others. But it must be remembered that in those comparative statements two most important variables are not taken into account. First, there is the varied rapidity of the local morbid process—a very considerable range of difference; and it is imaginable that the power of the intestinal flux to produce collapse may vary with the rapidity, rather than with the mere degree, in which it tends to inspissate the blood. Secondly, there is the varying susceptibility of the individual patient; and that this has range enough to account for very considerable differences of manifestation in the functions concerned in collapse will be evident to any one who has attentively studied the very kindred subject of febrile rigors. Indeed, the power of both the variables in question may be illustrated from that analogy; for all observers know how essentially the rapidity of the thermal rise is the determining condition for the rigor; and all equally know how one patient suffers rigor to the very verge of death from influences which would not appreciably disturb another one.

"In questioning the fact of a primary blood poisoning in cholera, I, of course, do not intend to deny that the blood *during* cholera is poisoned. From our earliest knowledge of the disease it has been on record that, when pregnant women have cholera, the intra-uterine offspring always invariably dies; and more recently, in proportion as the anatomy of the disease has got to be better studied, cases have accumulated, giving detailed evidence in support of an opinion which had from the first been entertained, that the infant in such cases dies of true choleraic infection. Waiving particular reference to earlier cases of this sort (for which see, for instance, Phœbus, 1833, op. cit. § 51,* and Buhl, 1856, in the famous Bavarian report) I may quote some statements made by Dr. Goldbaum in the report to which I have already referred. In the three last epidemics, he says he has carefully anatomized twenty-two such infants, and never failed to find appearances which, collectively, he deems characteristic of cholera. 'In the stomach and upper part of the small intestines always there was a fluid like rice-water, sometimes a thick mass, consisting of exfoliated bowel epithelium; the heart was always ecchymosed; at the back of the tongue there were swollen papillæ, as there are in greater degree in adult cholera corpses; and in the kidney the yellowish cortex contrasted strongly with the blood-holding medullary substance.' It may, I think, be assumed for certain that the death of the

* "Among the cases given in Phœbus's work is one where the infant was not actually born dead, but died an hour afterwards with all symptoms of the epidemic disease."

them suddenly rose in 1827, and progressively increased till 1831; and that diarrhoea has continued to go on gradually increasing in fatality since 1838. It proved fatal, *per se*, evidently as a variety of cholera, chiefly to young children and to old people, who did not so commonly exhibit the spasms of cholera (which are not essential to the disease), but died with nearly all the other symptoms of the malady. It proved fatal as a cause of death in other diseases. "It killed sick and dying men;" and now, at least, a belief prevails that cholera has become indigenous to this country; and assuming that it is produced by the action of a specific poison, how is it reproduced and propagated?

The chronological and geographical history of cholera has commonly been considered as affording *prima facie* evidence that, as regards this country, it is originally an exotic disease, the product of another climate, which yet has met with conditions favorable for its development and propagation in this country. It is now quite clear (as Dr. Macpherson has shown) that exactly the same disease as Indian cholera was known in India when the Portuguese went there first about the year 1500. The disease in India has had periods of increase and of decrease; but it was never diffused in that country so widely as it has been since 1817; nor had it ever wandered so extensively from India before. Whatever cholera we may have had in Europe in former times, few doubt that since 1817 it has always been carried out of India, or Persia, or Arabia, into other places.

Several explanations or theories have been proposed, to explain the occurrence of cholera in this country. According to Sir Ranald Martin, they may be referred to the following six heads:

1. That the disease spreads through *atmospheric influence or epidemic constitution*, by a succession of local outbreaks, and that the particular localities affected are determined by certain "*localizing conditions*," which are—*first*, all those well-known circumstances which render places insa-

fectus is death by cholera,* and that the fetus is infected through its blood. And since its blood is a mere derivative of the mother's blood, the fact seems to be beyond dispute that the mother's blood had cholera contagium in it. In relation to our main argument, however, the question is virtually unchanged. Is there any reason to suppose that the cholera contagium in the mother's blood was not a secondary product of disease—was not let into her circulation from the ferment-seething interior of her bowels? In this point of view the case may be usefully illustrated by another and closely kindred fact. Dr. Thudichum (see p. 477, *Ninth Report on Public Health*, 1867) has made the important observation, that sometimes in cholera the blood, like the rice-water of the intestinal canal, contains butyric acid. He does not believe that this poisonous product of fermentation is primarily formed in the blood; he believes that it is only to be found there when, after collapse, absorption from the bowels has recommenced, and when evidently the presence of that and other like matters in the blood can be interpreted as a fact of secondary infection from the bowels.¹²

* "I have been most anxious," says Mr. Simon, "if possible, to bring this assumption to the test of actual proof, by causing infection experiments to be made with the intestinal contents of such cases; and I have communicated with various persons on the subject. Unfortunately, the only fetus which came within our reach was that referred to in Dr. Thudichum's report. Its intestinal contents were given to Dr. Sandersen for the purpose, and were used by him in the intended manner, but with only a negative result. The time of year had unfortunately been reached when, as he has stated, all infection experiments failed; and therefore no conclusion whatever can be drawn from this one test of the fetal rice-water. The general evidence, however, seems fairly conclusive as to their nature."

lubrious ; and, *second*, a susceptibility to the disease in the inhabitants of such places, produced by the habitual respiration of an impure atmosphere.

2. That the cause of cholera is a morbid matter which undergoes increase only within the human body, and is propagated by means of emanations (or discharges) from the bodies of the sick ; in other words, simply by contagion in the most limited sense of the term.

3. It is believed that the poison of cholera is swallowed, and acts directly on the mucous membrane of the intestines, and is at the same time reproduced in the alimentary canal, and passes out much increased, with the discharges ; and that these discharges afterwards, in various ways, but chiefly by becoming mixed with the drinking-waters in rivers and wells, reach the alimentary canals of other persons, and produce the like disease in them.

4. Assuming that the cause of cholera is a morbid matter or poison, it is probably reproduced in the air, as well as within the bodies of those whom it affects, and that its effusion may be due to the agency of the atmosphere.

5. It is believed that the cholera poison is increased by a species of fermentation, or other mode of reproduction, in impure, damp and stagnant air ; and it is maintained that it nevertheless is distributed and diffused by means of human intercourse, being carried in ships and other vehicles, and even in the clothes, especially in the foul clothes of vagrants, and the accumulated baggage of armies.

6. It is assumed that the material causes of the disease may be increased and propagated in and by impure air, as well as in and by the human body.

"Germany and England," as Mr. Simon justly observes, "may claim between them the credit of having built up all the definite knowledge we yet possess regarding the pathology of cholera." Griesinger and Hirsch of Berlin, Wunderlich of Leipzig, Pettenkofer of Munich, Thomé of Cologne, and Klob of Vienna ; combined with Orton, Tytler, Parkes, Greenhow, Snow, Sutton, Martin, Macpherson, Buchanan of London, and Dr. Andrew Buchanan of Glasgow, Budd, Baly, Gull, Marshall, Radcliffe, Lauder Lindsay, Thudichum, Sanderson, Beale, Farr, and Simon, are those who have most of all contributed extensive and definite observation under varied circumstances and numerous epidemics in this country and in India.

The microscopy of the body in cholera, and especially of the stomach and intestines, has led in Germany to the rediscovery (by Drs. Thomé and Klob) of microscopical bodies like *fungi*, innumerable and vehemently multiplying, whereof swarms are shed with prolific and infective power in each characteristic evacuation of the sick. At the International Medical Conference on cholera, which met at Easter of 1867, at Weimar, and which Mr. Simon attended, Professors Hallier of Jena and De Bary of Halle (two of the leading mycologists of Germany) were associated with Dr. Thomé and Klob to make, in common with them, a statement and appreciation of the facts which had been observed, and which are in substance that— "Both observers find in cholera evacuations and in the intestinal mucus of the dead body definite organic structures, consisting of excessively fine granules, clustered more or less densely in the inter-

spaces of a jelly which surrounds them. The granules divide and subdivide themselves, to form beaded threads, which interlace in immense numbers into felted masses in the mucus. The further development of these organisms have been determined by Thomé and Hallier. By sowing or cultivating them, these observers have got, after some time, larger round cell-like bodies, which rapidly multiplied, and also abundant filamentous fungi (*cyllindrotetium*) on which grew cylindrical spores, capable of developing again to filaments" (*Ninth Report of Mr. Simon*, p. 31).

It is the peculiar property of some of the fungi to develop a viscid secretion by which they are enveloped, and the gelatinous mass with which the spores described by Klob is surrounded seems due to this development by the spores of fungi, and is not to be confounded with the intestinal mucus. This gelatinous mass forms with the spores the *Zooglaea termo* of Cohn; and in some instances the intestinal contents were entirely composed of this *bacterium* jelly.

In 1849 the question of fungi in cholera stools was repeatedly discussed on the repeated observation of several independent observers; but the objects then described do not seem capable of precise identification, or as being exactly the same as those now described. On the authority of Mr. Simon, it would appear that Boehm in 1838 describes "the whole extent of the intestine as teeming with a vegetation of *micro-fungi*; that innumerable round and oval, or more elongated corpuscles are to be found in all the vomit and dejections, as well as in the canal, sometimes single, sometimes two, three, four, or more, joined end to end, as links of a chain, and these chainlets sometimes branching; that such forms are held together in mucous floccules, and come best to light when *liquor potassæ* is used; that within the small intestine they are often so numerous that not the smallest specimen will fail to show numbers of roundish *fungic* forms amid the debris of epithelium" (*Über das vorkommen der Gährungskeime (Pilze) in Nahrung-Kanal der Cholera-Kranken*, quoted by Mr. Simon, *Ninth Report*, p. 518).

In the microscopic examinations of the "rice-water stools" of cholera in 1848 made by Dr. Parkes (whose observations were at the time confirmed by Drs. Sharpey and Jenner, and by Bowman, Hillman, Ellis, and Quekett), "peculiar corpuscles" were observed, and were variously named as such, or as "dark-yellow granules," or as "organic corpuscles (about the size of the pale corpuscles of the blood), finely granular on the surface, and containing from six to twelve dark-yellow or black granules." Without being able to state what these "peculiar corpuscles" are, Dr. Parkes goes on to show that they are not mucous, but that their existence and development is confined to the deep algide period—that they are not seen in the premonitory diarrhoea, nor after the algide stage, and disappear when the pulse and the warmth of the surface are returning. They co-exist in their greatest perfection with the purest type of the cholera fluid (*London Journal of Medicine*, 1849, p. 144, *et seq.*). Thus Dr. Parkes, and those observers who aided him, recognized certain "peculiar corpuscles or granules" in the rice-water stools of cholera; but neither of them suspected the peculiar corpuscles or granules to be

of vegetable or fungoid origin. They appeared then to Dr. Parkes to be "but modifications of the same substance, namely, fibrine." He, however, recognized "vibriones in great numbers, and two or three oval transparent bodies placed end to end. When the stool was kept, these fungi increased in numbers" (Ob. 6). In the descriptions and plates of Klob, Thomé, and Hallier, the corpuscles and granular bodies which they figure are now recognized by Dr. Parkes as similar to those which so attracted his attention in 1849. He again noticed these bodies in 1865 and 1866, when they vividly recalled the previous observations to his mind. Dr. Parkes concludes from these and his own observations that this fungus development really exists, and invariably in cholera dejections. He found them in every stool which he examined in 1849 and in 1854; and he again saw them in 1865 and 1866. He is familiar with all the forms described by Klob and Thomé; and believes them universal in cholera stools; and they form the major part of the white flocculi of the true "rice-water stools" (*Report on Progress of Hygiene in Army Med. Dep. Reports for 1865*). In 1854, Dr. Lauder Lindsay also recognized large bodies which he named "gonidic," from their resemblance to the gonidia of the lichens. They appear quite globular, usually larger than pus-corpuscles; have a distinct wall, colorless and transparent; frequently a distinct central nucleus, also colorless, round which are aggregated a number of rounded granules of a bright greenish-yellow or orange color, resembling the chlorophylle grains in the cells of plants. These bodies have occurred in greater or less abundance in the evacuations of *all* the cholera patients under Dr. Lindsay's charge; and they pass through the digestive apparatus both of man and the dog apparently without change (*Edin. Med. and Surg. Jour.*, 1854).

To say that these fungi are the cause of cholera would as yet be premature; nevertheless their existence is capable of accounting for many of the phenomena of cholera, and especially its spread. The poisonous properties of fungi are well known, so that, apart altogether from the physical influence of such spores, by their development in the intestines, the products of their growth may be eminently poisonous and deadly. In this direction the cultivation experiments may yet be carried.

Professor E. Hallier, of Jena, whose reputation as a fungologist is not surpassed, has made numerous experiments by the cultivation of these spores; and from an able summary of his paper by Dr. Buchanan (*Ninth Report to Privy Council*, p. 512), it appears that his observations were made on the stools of a person ill with cholera at Berlin in 1866, and on the stools and vomita of a cholera patient at Eberfeld in 1867. The characteristic vegetable elements consisted of a fine fungiform matter which floated, and of more highly developed spore-cysts which sank to the bottom (Figs. 1 to 4. of engraved Plate). These spore-cysts were yellow or brownish bodies, consisting of a pale membrane inclosing highly refracting colored spores. The cyst-wall undergoes a series of changes, ending in its rupture or solution, when the spores become free. The spores then, by progressive (*uniparous*) partition (a process which may begin before

they leave the cyst), resolve into very small cells, grouped into balls and heaps, which Hallier calls "colonies of micrococcus." The small cells constitute the fungiform matter seen in the evacuations, and they attach themselves to any bodies there may be in the stool—to remnants of animal or vegetable food, to epithelium cells, or to oil-globules, when these and all nitrogenous matters to which these fungi cells fix themselves become of a dirty aspect and lose their structure.

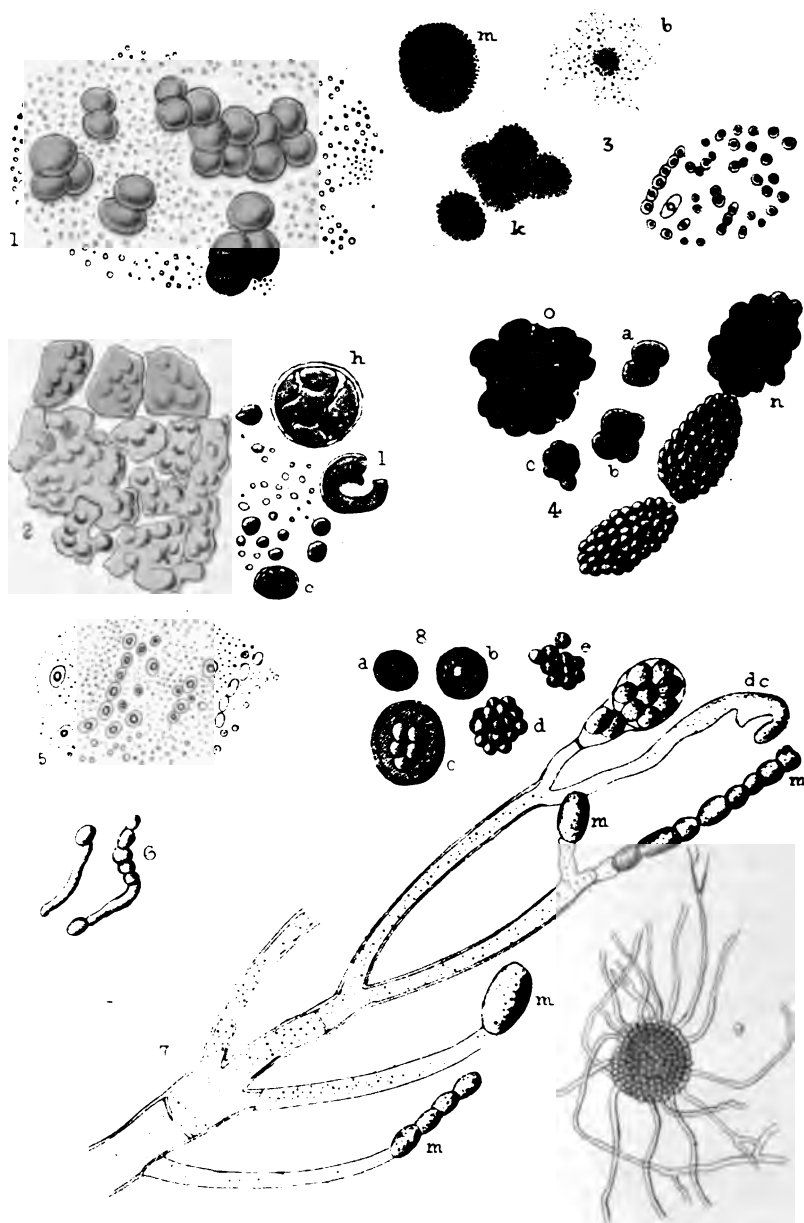
Besides these two elements (the spores and minute cells), torula-like bodies (Fig. 3, *c*) were found in smaller number, and were shown by cultivation to develop from the micrococcus cells. The formation of these cells (which occur singly or in rows) marks a step towards a higher development of the micrococcus—towards the production of *oidium** forms of fungi.

In the stool and vomit from Eberfeld, the cysts and colonies of fungi were found in smaller proportion, and the free micrococcus cells more abundantly, and epithelium was seen in which the process of invasion by the micrococcus could be watched. The little cells fastened themselves upon the epithelium, and increased in size as the epithelial elements wasted. The parasitic cells of fungi always grow at the expense of any nitrogenous organic substance which they attack; and this is well seen in the fungus foot disease (*Mycetoma*) of India. (See end of Vol. I.)

To learn the development and ultimate form, so as to identify the fungus, Hallier had recourse to the artificial cultivation of the spores, on several substances on which *fungi* are known to increase, multiply, and grow, such as in sugar, starch, paste, flesh, and the like (Figs. 5 to 8 of engraved Plate), in the same way that our distinguished fungologist, the Rev. M. J. Berkeley, made out the nature of *Mycetoma*. In sugar, Hallier, conducting the growth at a temperature of 68°–88° Fahr., succeeded in growing from the spore-elements a long pale filament containing granular plasma, and divided by septa; from this elongated processes branched off, and the whole formed a structure greatly resembling the *oidium lactis*. This *oidium* plant bore at the ends of its branches bulbs (*macroconidia*, Fig. 7, *m*), either single or in shorter or longer series: if single, generally larger, and apt to develop *muror* forms; but if in series, more apt to develop *penicillium* forms. On the ninth day of the experiment some of the branches bore a cyst containing spores, pale and weakly. Only once did a well-developed colored spore-containing cysts make its appearance; and Hallier's experience led him to connect the absence of such cysts with the absence of nitrogenous matter from the soil in which the fungus was growing.

* The *oidium* fungi belong to the order *mucedines*—the "blue moulds"—having flocci very short, producing a moniliform string of spores by tomiparous division. In temperate climates they grow on damp paper, dead wood, decayed fruit (grapes, oranges), on *porrigio lupinosa*, on honeycomb, on nettles, ground ivy, and on plants infested with *ergot*. They belong to the family *Hypomyces*, or "thready fungi," the characteristics of which are filamentous, with fertile naked threads, for the most part free, and bearing the spores at their apices. (See Berkeley's *Outlines of British Fungology*, pages 337 and 350.)

FUNGI AND THEIR DEVELOPMENT BY CULTIVATION.
 FROM THE "RICE-WATER-LIKE-STOOLS" OF MALIGNANT CHOLERA
 (AFTER DR ERNST HALLIER)



A variation of the experiment, by providing the fungus with starch paste, produced scarcely different forms; but a more interesting result came of the addition of a small quantity of tartrate of ammonia to the paste. For the first few days of this experiment nothing but *micrococcus* cells, and chains of similar elements resembling *leptothrix*,* were seen; but about the fifth day there appeared a small brown speck in the paste at some little distance from the surface, at a spot where the reaction was alkaline. This brown spot gave to the microscope colored forms, filaments bearing *macroconidia*, single and in series (Fig. 7), and some of them also bearing bunches of spores, or well-developed cysts containing spores, and greatly resembling the cysts found in the original stools. Upon the occurrence of an acid reaction in the paste the growth of these bodies ceased.

The spores of the fungi were further grown upon muscular tissue immersed in sugar solution. The *micrococcus* cells were seen enlarging and budding, and developed the usual *oidium plant* with *conidia* at the ends of its branches. Shortly after numerous cysts appeared, which went through the same changes as the cysts in the original stool. The muscular fibres were invaded and decomposed by the *micrococcus*, just as the intestinal epithelium had been in the stool.†

A subsequent observation made with cholera fungi grown in Hallier's isolation apparatus, upon paste which had been boiled with tartrate of ammonia, gave highly developed cyst-formations

* These belong to the family *Coniomycetes*, or "dust-like fungi," of which the spores are the prominent feature, and not the threads (as in *Hyphomycetes*), the spores being either solitary or concatenated, produced on the tips of generally short threads, which are either naked or contained in a perithecium, or compacted into a gelatinous mass. *Rust* and *mildew* are examples of this family. *Leptothrix* consists of the mycelial filaments of mildew fungi, abounding in foul water in flocculent masses.

† EXPLANATION OF THE ENGRAVED PLATE IN ILLUSTRATION OF PROFESSOR HALLIER'S EXPERIMENTS ON THE FUNGUS FOUND IN THE "RICE-WATER" STOOLS OF CHOLERA (p. 589 to 592 of text).

Figs. 1 to 4.—Vegetable Growths, of the Nature of Fungi, seen in the Cholera Rice-water Stools: Berlin, 1866.

1. Groups of swollen gelatinous spores in the act of forming *micrococcus* by repeated divisions of the nucleus. The spores are surrounded by *micrococcus* cells.

2. Gelatinous cysts swollen and breaking up. (c.) Small cyst with clear spores; (l.) gelatinous cyst; (h.) wall of cyst subsequent to discharge of the spores.

3. Yeast formations. (m.) A large colony of *micrococcus* corresponding with a spore; (b.) a large colony of *micrococcus* about to break up; (k.) a group of colonies originating from the spores of a cyst; (e.) *torula*-like cells grown from swollen and enlarged *micrococcus*.

4. (n.) Several cysts connected; (o.) a large globular cyst discharging its spores; (a.) semi-divided spore, showing the commencing formation of *micrococcus*; (b.) spore quartered; (c.) groups of spores from a small cyst.

Figs. 5 to 8.—Products of "Cultivation," No 1: Berlin Rice-water Stool of 1866, with Sugar Solution.

5. *Micrococcus* and *torula* formations.

6. Germination of *torula* cells.

7. Filamentous termination of a healthy germ. (m) *Macroconidia*; (c.) cysts; (d.c.) degenerated cysts.

8. Completely formed cyst with spores, one of which is just about to germinate, (k.)

Fig. 9.—(a.) Commencement of proliferation.

with the contained spores in a state of actual germination, and pushing their processes through the cyst-wall. This is the exact counterpart of a form known as *Urocystis occulta*, found in the tissues of cereals; and a very similar form, *Urocystis intestinalis*, is found in diphtheritic diseases of the intestine. The observations of cysts apart from filamentous growth, as they were in this experiment, is particularly instructive, as it is under somewhat similar conditions that the development of the cysts, also apart from filamentous growth, takes place in the intestine of cholera patients. (Fig. 9, a, commencement of proliferation.)

Twenty-two such experiments were made with the cholera stools, and results consistent with those above described were obtained. Hallier's experience thus enabled him positively to prevent, and to know that he had prevented, the accidental entrance of any atmospheric fungus into his cultivation experiments. He proved the "peculiar corpuscles" to be spores of a fungus, and to belong to a species which comprises forms of *Penicillium** *crustaceum*, *Mucor*† *ramosus*, *Tilletia*‡ and *Achlya*§ which are four different forms or developments of the *Macroconidia* in the same *oidium-like* fungus. But in the actual rice-water stool it is none of these four developments, but a fifth, which systematists would place under the group of *Urocystis*.|| Hallier has obtained in the course of his mycological studies *oidium* plants exhibiting the other forms of growth, but he has never produced by artificial cultivation cysts of *Urocystis* on them, except from these cholera stools. Thus, in the course of innumerable observations on milk *oidium*, where *penicillium* and *mucor* were met with, nothing resembling the cyst forms was ever seen. From this and other considerations, Hallier infers that this form is not indigenous to Germany, and that it has travelled with cholera from India. In the next place, he cannot think that the original *habitat* of the fungus should be the human intestine, which is under much the same conditions in India as in Europe; but he sees in the high temperature of the intestine a condition capable of maintaining this fungus in activity, once it is introduced (as it may be by direct intercourse with India). A similar high temperature, as provided by the mean climate of India, and by the extreme summer climate of Europe, also furnishes the condition requisite for

* *Penicillium*.—Hyphomycetous fungi, the commonest constituent of green and blue mould, a form which grows in all kinds of decaying substances, but especially in semi-fluid matters.

† *Mucor*.—Physomycetous fungi, bearing vesicles containing indefinite sporidia, the common mould of paste and of decaying fruit.

‡ *Tilletia*.—Coniomycetous fungi, the "bunt" of various corn grains, especially the *Dhoeira* corn of the tropics and the *maize* of temperate regions.

§ *Achlya*.—An aquatic form of mucorinous fungi—parasitic on the bodies of dead flies in water, fish, frogs, and decaying plants. It appears as colorless filamentous tufts enveloped in a gelatinous cloud.

|| *Urocystis*, of Eastern habit, seem to belong to the forms of *Ustilago* of temperate regions, of the order *Pucciniae* and family *Coniomycetes*. The spores of this order are simple, springing from delicate threads, or produced in the forms of closely packed cells, which ultimately break up into a powdery mass. The spores produce in germination secondary spores, and the plant is parasitic on living plants, and is deeply seated. *Ustilago* yields the "smuts" on corns and grasses of this country.

the development of the fungus outside the body. Thus in summer, and in summer only, in European latitudes could the fungus find in earth and night-soil the necessary temperature for its increase. The conditions for the production of Urocystis appear by the experiments to be, not high temperature only, but also a copious supply of nitrogenous with some hydro-carbonous nutriment, and a high degree of moisture. But besides these conditions the reaction of the fluid was found to be important, and this again to be dependent on the nitrogenous elements of it.

Hallier points out that the home of all penicillium-bearing fungi is Asia. The tilletia-bearing form occurs only on wheat, which is a plant imported from Asia. Hence he infers a further probability that the cholera fungus, which appears to be another development of the same species, is also originally Asiatic.

Further observations as to the precise effects of temperature confirmed the foregoing deductions. Fungus, while developing Penicillium only at ordinary temperatures, was grown upon appropriate nitrogenous soils at a temperature of 88° to 110° Fahr., and (when other circumstances were favorable) a development of cyst-forms took place from the budding of the Penicillium, precisely like the forms met with in the stool. A piece of intestine exposed to the action of the cholera fungus at this temperature got its epithelial elements rapidly destroyed by micrococcus. Converse experiments with low temperatures showed that the characteristic cyst-forms of cholera stools were not produced upon materials that were kept below 54° Fahr. The inference is therefore confidently drawn, that if the fungus be indeed the contagious material of cholera, cholera cannot maintain itself permanently in our latitudes. Other conditions under which the fungus did not grow were,—(1.) A temperature over 144° Fahr.; (2.) Sulphate of iron in concentrated solution; (3.) Carbolic acid (not the most potent agent of its kind in these experiments); (4.) Permanganate of potash; (5.) Wine (from its acidity, probably) and strong alcohol. Quinine had some influence, opium none, in preventing the destruction of animal tissue by the micrococcus. Of all experiments made to determine the power of chemical agents upon the fungus, chief success was obtained by the free acidification of the fluid. This is confirmatory of Pettenkofer's views upon the disinfection by acids of substances infected by cholera poison. Whenever the fungus grew in acid solutions it showed no cysts and no micrococcus, only penicillium and cognate forms. For fungus destruction on the large scale Hallier would give preference to sulphate of iron. But he insists particularly on the destruction of each individual stool before mixing it with other night-soil, and of course urges the systematic removal of all such matters to the field.

Professor Hallier's inquiry is next concerned with the circumstances under which the cholera fungus, indigenous to Asia, and only travelling into northern latitudes in the bowels of cholera patients, grows in its native soil of India. He recalls the fact that other forms of the fungus under consideration are peculiar to cereal plants, and that the Urocystis, with its characteristic cysts, inhabits

the delicate and highly nitrogenized tissues of grasses; and he asks whether the cholera-cysts may not also in their native soil be parasites to some graminaceous plant, just as the form *Tilletia*, which can exist in a European climate, is a parasite upon the imported cereal, wheat, which acclimatizes itself in these latitudes.

Herein the circumstance assumes a peculiar importance, that at their first acquaintance with cholera English physicians in India named it "rice disease" (*Morbus oryzeus*, Tytler), and connected it with a diseased condition of the rice plant. Examination into the existence of a similar fungus attacking rice in India must of course be undertaken by inquiries in that country; and on the best authority (that of the Rev. M. J. Berkeley) we know nothing of the microscopic fungi of India; but Hallier makes a notable contribution to this aspect of the question by his experiments. He planted rice under conditions of heat and moisture as nearly as could be obtained like those of Asiatic rice-fields, and he watered these plants with the stools and vomita whose investigation had occupied the earlier parts of his paper. He obtained in every one of these experiments positive results. Carefully taking out his little rice plants in an early stage of their growth, he made longitudinal sections of them, and found fungus threads in great numbers perforating the epidermis of the plant in several places above the junction of the rootlets. The cells, as well as the intercellular spaces of the tissue, were invaded, and the delicate plasma of the cells was shrivelled and coarsely granular; and by the aid of glycerine a multitude of cryptococcus cells were seen, and had the same characters as when known *Urocystis* grows within cereal plants. Here his investigation ended, without proof having yet been obtained of the identity of the parasitic fungus with the cyst-bearing plant, but with the important result that a form of the same type, at any rate, could be produced in rice during its growth, when watered with the cholera evacuations (BUCHANAN, *l. c.*)

All the theories now given agree in assigning an Eastern origin to the poison of cholera, which is believed to have either reached this country by means of direct human intercourse, as by fomites or individual contagion; or the poison itself is assumed to be migratory, and to have come hither by a kind of wave-like extension from India.* All of the suppositions (with the exception of the *third*, propounded originally by Dr. Snow) consider the existence of certain local conditions, or of a predisposition in the inhabitants of infected districts, as usually necessary to give strength and vitality to the poison. It is also to be observed that, while each of the six opinions (seven, if we include the fungoid one) or theories just noticed is apparently supported by a large amount of evidence,

* Our knowledge of the progress of cholera in Great Britain was originally contained in an official report presented by the Commissioners to His Majesty King William IV, of which only one copy existed. This was rescued from oblivion by Sir James Clark, aided by the Royal Librarian, who found it in a drawer, buried among a heterogeneous mass of papers. It is now appended to Dr. Graves's *Report on the Progress of Cholera*.

direct and circumstantial, each is also equally opposed by a "considerable number of obstinate facts." All of them, however, agree in two main points—namely, that cholera is induced by a special poison, and that this poison is of foreign extraction.

But, on the other hand, there are good grounds for believing that cholera has not in the present century for the first time appeared in this country, and extended itself over the greater portion of the habitable globe. The "*cholera morbus*" of Sydenham, prevalent in his time, and the "*griping in the guts*," or "*plague in the guts*," as recorded in the mortality bills of 1665, and described by Willis, and subsequently by Dr. W. Heberden, Jr., do not seem to differ in their essential phenomena from the disease supposed to be imported into this country from the East; although some believe that these descriptions of disease refer to dysentery, and not to cholera.

There are also abundant facts which seem to show that, under a different name, cholera was one of the most fatal epidemics by which the population of London was formerly afflicted. And there is no doubt that cholera, like every other epidemic disease, varies in its type, as it does in severity; for, if it is conceded that the diarrhoea so prevalent during an epidemic of cholera arises from the same cause, and is, in fact, the same disease in a different degree of intensity, as Orton showed in 1832, "there is as much variety in the aspect and symptoms of cholera as of scarlet fever; between the malignant cases of which and the extremely mild ones there is so vast a difference."

The principal differences shown by Mr. Radcliffe between the recent epidemic (1866) of cholera in Europe and those of former years are, (1.) For the first time in the history of the disease, Europe was invaded from the south. In 1829–32 and 1845–48 the disease spread from Persia to Russia, and thence along the Danube into Central Europe; and in Britain the towns first attacked on all previous occasions were sea-ports on the east coast. (2.) A remarkable feature in the epidemic of 1866 was its rapid and great extension along the coast line as compared with its slight and sluggish penetration inland. The central districts of Europe escaped altogether during 1865, except that there was an isolated outbreak in Saxony. (3.) The progress of the disease was much more rapid than in former epidemics. In 1829 cholera took fifteen months from the time of its entrance into Europe to reach Great Britain; two years, less one month, to arrive on the North American coast. In 1848 its diffusion occupied nearly the same period of time. In 1866 the disease had in less than five months spread from Alexandria to the coasts of the Euxine, and even to the western hemisphere. A strong point in favor of the view that in 1866 cholera was introduced from the Mediterranean by ships coming thence is furnished by the fact that Southampton was the only port at which ships arrived having had cholera deaths on board shortly before reaching England. Moreover, it had been predicted that the disease would enter the country by Southampton, and not, as before, by towns on the east coast; and the fulfilment of the prophecy will seem to many, as Dr. Parkes

observes, sufficient evidence that the outbreak at Southampton arose in this way (*Sydenham Society Biennial Retrospect*, 1867, p. 494). (4.) This swift propagation of the epidemic does not appear to have been dependent on any peculiar virulence of the disease.

The evidences of importation or transmission of the disease in this country in 1866, by human intercourse, were sufficiently abundant, and as demonstrative as possible consistent with the nature of such evidence; and there can be no doubt, as Mr. Simon concludes, that if a quarantine could be conducted with the extreme rigor and precision of a chemical experiment, cholera could be kept out of any part of Europe wherever such extremely difficult conditions could be absolutely fulfilled. England seems to have been infected in 1866 at many different points of invasion, and from many different directions, almost at the same time, as the following details by Mr. Simon will show:

"On the 28th of April a first case was reported from Bristol—that of a trader who had arrived there sick from Rotterdam.

"On 15th May telegrams from Liverpool and Birkenhead reported that the disease was prevailing on board certain vessels in the Mersey, among German and Dutch emigrants, who, with a view to crossing the Atlantic for New York, had come in flocks, travelling rapidly from the Continent, often from infected parts of it, by way of Hull, Grimsby, and other of our northeastern ports, and had now fallen ill at their port of embarkation. Much alarm was occasioned by this outbreak; the more as new arrivals of the same sort were occurring from day to day. The outbreak, so far as England was concerned, soon came to an end; but the subsequent progress of the emigrants was unfortunately not unattended by cholera. Indeed, in several cases, vessels such as the above, leaving in apparent health, suffered during their voyage cholera deaths among their passengers and crew, and were of course very dangerous arrivals for their port of destination.

"Within the next few days after the 15th, my Lords of the Privy Council were apprised of the first two cases of what afterwards became a serious epidemic at Swansea; and single cases in various other parts of the country were also notified to them. Anxiety became general in the country; and there was much correspondence with local authorities, often on precautions to be taken against the disease, or provisions to be made for treating it, and often on questions of jurisdiction and responsibility.

"On 15th June the Peninsular and Oriental Company's steamship 'Poonah' arrived at Southampton with a case of cholera on board, and several other cases had appeared in the town. On the 29th two deaths were reported by telegram to have happened at Goole, whither clearly the disease had been imported from Antwerp. On the 30th three deaths were reported to have occurred at Northwich in Cheshire, and on the same day a case of cholera occurred at Shields on board the 'Clio,' from Hamburg. On 3d July a case was reported to have happened at Harwich on board the 'Redstart,' from Brussels; and on the same day from Brixham, the death of the captain of a coasting vessel was reported.

"A serious extension of the disease was imminent. Reports of new centres of infection became more and more frequent; and on 14th July, the time had come for putting the Diseases Prevention Act in force throughout the whole of England and Wales, by which ample powers of

medical relief (not restricted to paupers) were exercisable by local authorities throughout the country.

"On 18th July, from Poplar, the first cholera death in the metropolis was reported. Two days afterwards there was already an alarming proportion of cholera cases in parts of East London; and on the 21st the secretary of the London Hospital reported that the resources of that most useful institution were being overtasked by such claims for admission as attested a very terrible epidemic of cholera."

At the same time diarrhœa of so severe a form as to be called "choleraic" preceded the epidemic outbreak in 1865 and 1866; and if it is found impossible to disentangle at the beginning of an outbreak, cases of a *quasi* epidemic character from those of a *true* epidemic character, and to shut out absolutely a theory of the development of epidemic cholera by gradation out of quasi epidemic or severe diarrhœa, it is equally impossible to set aside the fact of exposure of the metropolis (and busy ports of embarkation and debarkation, like Southampton and Hull and Liverpool) to continuous transmission of the epidemic malady from the early autumn of 1865 to the early summer of 1866 (RADCLIFFE). The apparently distinct outbreaks in several towns and localities, however isolated, must all be regarded as forming parts of one general epidemic; and the histories of them, so ably set forth in the various reports collected together by Mr. Simon, Dr. Parkes, and others, compel the conclusion that the chief agents in the dissemination of the epidemic of 1866 have been the sick from the malady in its slighter, as well as more marked and characteristic forms—a conclusion which has been adopted absolutely of epidemic cholera by the International Sanitary Conference which met at Constantinople to consider the question of the preservation of Europe from this pestilence. The history of the epidemic in its entirety in this country points to the transmission of the disease to the metropolis and other ports from localities previously visited by it in Western Europe. Mr. Simon's dictum that "*contagious currents on the Continent of Europe must be deemed virtually current in England,*" is to be accepted as an axiom in State Medicine, notwithstanding that links of transmission may fail to be discovered (*Ninth Report*, p. 288). The testimony of Dr. Macpherson also is to the effect that, whatever cholera we have had in Europe in former times, since 1817 at least it has been always carried out of India, or Persia, or Arabia, to other places.

"The outbreak of cholera in the metropolis in 1866 cannot well be considered apart from the wide diffusion of the disease on the Continent of Europe during 1865 and 1866. It is inextricably linked, both chronologically and etiologically, with that rapid dissemination of the malady which in May of the former year, commencing at the most sacred city of Mohammedanism, Mecca, extended to Egypt, and thence, before the close of the summer, to many places on the eastern and southern coasts of Europe, and in the basin of the Mediterranean. During the autumn the epidemic spread largely in the south of France and in Spain, appeared at Altenburg in Saxony (where it was introduced from Odessa),* and ex-

* *Die indische Cholera in Sachsen*, 1865; Dr. Rudolf Günther, p. 9.

tended to several neighboring towns, broke out with severity in Paris, and infected slightly our own coast at Southampton. From the 24th September to the 4th November thirty-five individuals succumbed to the disease in the last-named sea-port town; and from the 28th September to the 31st October nine deaths occurred from cholera (an offshoot of the Southampton outbreak) at Theydon Bois, in Essex, a hamlet lying about eleven miles in a direct line N.N.E. from Bow bridge. As the winter of 1865-66 advanced, the epidemic extended to Northwestern France, chiefly affecting the departments of Finisterre, Morbihan, and Côtes du Nord; and throughout the cold season it manifested more or less activity along the opposite coast of the Channel. In the northeast the department of the Vosges received the infection. With the increasing spring the disease became more rapidly disseminated. In several localities of Belgium and Holland it early showed itself. As the summer grew, and its mid-season approached, the diffusiveness of the epidemic augmented largely. The malady reappeared in several cities and towns of Eastern, Southern and Western Europe which had suffered from it the previous year; it spread generally throughout the provinces of Belgium and Holland; and extended widely in Prussia, Central Europe, and European Russia.

"The epidemic broke out in Rotterdam prior to the 21st April; in the port of Antwerp on the 19th May; in Stettin before the 2d June; in St. Petersburg on the 26th June, or somewhat earlier; in Memel before the 10th July; and in Dantzic before the 12th of the same month" (*Ninth Report to Privy Council*, by Mr. Simon).

But other and occasional circumstances are required to give energy to the development of the poison of cholera. These may be described under the two heads of "*meteorological conditions*" and "*localizing causes*."

(1.) *Meteorological Conditions*.—Of the first of these, temperature appears to have some marked influence. The average temperature of 1846, in which the mortality occasioned by diarrhoea, cholera, and dysentery was very large, was 4° higher than that of 1845, and 3° above the average of the six preceding years, and the fall of average temperature was accompanied by a corresponding fall of mortality from the choleraic and flux diseases. Dr. Barton, of New Orleans, states that cholera always coexists there with an east or southeast wind. A temperature above 70° Fahr., increased as the disease attained its maximum, a dew-point of from 60° to 70° , and a barometric elevation of over 30° . The maximum barometer occurred on November 18, 1853, and was 30.46° (a very unusual height), soon after which cholera broke out. During December the wind continued from the east, north, and northeast; the maximum barometer was 30.48° on the 2d, when the cholera was at its height, and declined to its minimum, 20.57° , on the 30th. The cholera ceased soon after the middle of the month.

The first epidemic of cholera in this country, during the present century, began in the north of England, in October and November, 1831. The preceding summer was unusually fine, the nights being warmer, in proportion, than the days. In November, December, and January, the atmosphere was observed by many independent observers, both on land and sea, to be *singularly stagnant, unusually still, close, and hot*, so that it was impossible to ventilate even large

houses, in which no change of air "seemed to take place for almost a week together." According to the delicate and accurate observations of Mr. Glaisher, the meteorological phenomena of the three visitations of 1832, 1848, and 1854, appear to have been remarkably similar (excepting as to temperature). Indian medical officers, and those of the Black Sea fleet, give similar accounts as to the meteorological phenomena which attended the outbreaks of cholera, in their experience.

The chief meteorological phenomena of the epidemic period of 1865-66 have been summed up by Mr. Glaisher,* and a comparison instituted between them and those occurring during the previous cholera outbreaks. The contrast is remarkable. The visitations of 1832, 1848, and 1854 were coincident with great atmospheric pressure, high temperature (except in 1832†), small diurnal range, (owing mostly to high night temperature), deficiency of rain, very little wind (and comparative stagnation of atmosphere and prevalent mist), a deficiency of electricity (indicated by the few electrical disturbances), and in 1854 "the presence of a remarkable blue mist," which prevailed night and day, and total absence of ozone.

During the three months of principal prevalence of the outbreak of 1866 in the metropolis (July, August, and September), the atmospheric pressure was remarkably low. From the 26th of July to the end of the quarter the barometer, reading at the height of 160 feet, never reached the point of 30 inches—"a most rare occurrence," as Mr. Glaisher writes.

The temperature of the air was low night and day, except in September, when the nights were warm. The daily range of temperature was small, "chiefly owing to low day temperature, particularly in August, and to a somewhat less degree in September, but the range in September was still further lessened by the high temperature of its nights." There was an abundance of rain, and the air was in almost constant motion, "frequently blowing a much heavier gale than usual at this season of the year." "Nearly all the circumstances," Mr. Glaisher observes, "are directly opposite to those mentioned above as being present at the previous visitations of cholera, and have probably aided in checking its wider extension." He adds, "One of the most remarkable atmospheric phenomena during the past quarter has been the prevalence of a peculiar blue mist, first seen by myself on 30th July, but which had been remarked by other observers in the preceding week. This blue mist since that time has been generally present. On some days no trace of the mist has been visible, and on other days it has been seen for parts of a day only. It has extended from Aberdeen to the Isle of Wight, and of the same tint of blue everywhere. This mist increased in intensity when viewed through a telescope; usually no mist can be seen when thus viewed; it increased in density during the fall of rain; usually mist rises after the fall of rain. Its density did not decrease when the wind was blowing moderately strong, but did decrease when a gale was blowing, but increased again on its subsidence. I do not know the nature of this blue

* *Quarterly Return of the Registrar-General, July—September, 1866*, p. 18.

† *Appendix to Report of Committee for Scientific Inquiries—Cholera Epidemic, 1854*, p. 114.

influence; but the fact of its presence not having been noticed since the cholera period of 1854 till now, points out a possible connection; but, independently of this, it is of high meteorological interest."

Mr. Glaisher's observations are restricted to the September quarter; but Mr. Radcliffe extended his observations over the whole period of the outbreak in London, and especially calls attention to *two* points. The first of these is the probable effects of certain excessive variations in the temperature of the air upon the early, sudden, and large development of the outbreak; the second, the relationship of the fluctuations of the outbreak during its decline to variations of temperature; also to the deficiency of ozone during the four weeks in which cholera became active and the outbreak attained its greatest development.

The initial activity and rapid development of the outbreak in London of 1866 was preceded and accompanied by an excessive range of temperature. The mean temperature of the week ending the 30th June, in which the earliest cases of cholera occurred, was 4.8° above the mean of the same week on an average of 50 years. During the next week the mean temperature fell 5.1° below this average; but in the following week the mean was in excess 6.3° . In the first third of these weeks the range of temperature was 24.0° ; and in the second 17.5° .

It is not improbable that the great range of temperature in the last week of June and the second week of July was influential to some extent in causing the sudden development of the epidemic in other districts than the East of London during the third and fourth weeks of July and first week of August.

The relationship here suggested between the temperature and the development of the epidemic in the districts referred to is supported by the correspondence between the lagging of the epidemic during its decline and certain sudden elevations of the temperature above the mean. The rate of decline of the outbreak was much slower after the third week of fall than in previous epidemics. The epidemic lagged, in fact, and this lagging first followed and partly accompanied a three-weeks elevation of the mean temperature slightly above the average, after a four-weeks persistent fall beneath it. And it may be here observed that of the four weeks during which the mortality of the outbreak was greatest, and in each of which the temperature fell below the average, two of the weeks were weeks of rapid decline of the disease.

An augmentation of the outbreak in the last week of September and first and second weeks of August, during which the mortality in the metropolis, exclusive of the East Districts, reached its maximum, occurred contemporaneously with a three-weeks elevation of the temperature above the average; and the subsequent fall of the epidemic, near the commencement of winter, contemporaneously with a temperature maintained above the average, was sluggish. Among the facts about cholera, in India, none are more clearly made out than its prevalence in different places at particular sea-

sons. It is very plainly influenced most by the combination of influences known as "season" (MACPHERSON).

Such meteorological conditions have a marked tendency to *favor the chemical decomposition of organic substances, and to render the season defective in those atmospheric changes which, by decomposing and dispersing into space the products of decomposition, renew the purity of the air.* "The effect of temperature upon the Thames water is very remarkable in tainting the surrounding air, and is exhibited in the well-known fact that diarrhœa and summer cholera become prevalent among the inhabitants along the banks of the Thames after the temperature of the river has attained to 60°, and as the water declines from this temperature, so do these diseases in its vicinity." In Europe all the great epidemics have occurred in times of prolonged drought; and the dissemination or dispersion of the disease is very closely related to rainfall, as Dr. W. Budd, of Bristol, has shown. By diluting the poison, and by giving rise to floods which rapidly sweep it beyond the inhabited area, rain seems to have a powerful influence in checking the disease. But to have this effect the rainfall must be heavy and continuous—while, on the contrary, light and intermittent rains favor its spread.

The general result of all such observations is, "that whilst cholera may prevail within a considerable range of temperature, a moderately elevated one is most suitable for its development and propagation; and this, accompanied by a still, stagnant, and peculiarly oppressive condition of the atmosphere (more oppressive than the elevation of the thermometer can account for) and a moderate amount of moisture." With regard to the apparent anomaly as to temperature in the case of its outbreak in Moscow, and in the northern countries of Europe, such as in Sweden and Norway, it must be remembered that the internal atmosphere of the Russian, Swedish, and Norwegian houses is maintained at a high elevation during the winter months by means of stoves. It must be remembered, too, that the water used by the Russians in winter is often got from the melting of snow in the vicinity of the houses, and which snow is generally exposed to the reception of various excreta from the houses, just as the surface of the soil would be exposed. Hence the facilities for its spread in Russian hamlets.

Although meteorological statements appear to be a mass of confusion, from which we can scarcely deduce a single general principle; yet we know that organic germs, and seeds of various kinds, are capable of preservation under the most different and variable meteorological conditions; and also that particles or germs most microscopically minute are capable of actual demonstration in the air we breathe, as already stated at page 420. See also a most interesting and very suggestive paper on "*Germinal Matter and the Contact Theory*," by James Morris, M.D., Fellow of University College, London, 2d edition, November, 1867.

(2.) *Local causes.*—But, in order to give character and energy to the development of cholera, there are other conditions required besides those meteorological phenomena just noticed. These other conditions are described by Dr. Barton as the "*terrene element*," and

correspond with what in this country have been termed the "*localizing causes*" of cholera.

That some local circumstances play a very important part in the evolution of cholera, is evident from the following facts:

1. An analysis of the history of cholera epidemics shows that they are most frequently made up of a succession of partial local outbreaks, not only in different districts, but even in the same place.

2. The pestilence has also been observed to linger in some few favorite haunts throughout the entire course of an epidemic; and that, now and then, after visiting a place at the commencement of an epidemic, it has returned to it again, after an interval of complete immunity, before its close.

3. That some places escape an epidemic visitation at the very period when others in the immediate vicinity are suffering severely from its presence, the meteorological influences being the same. Even in the same town, whilst the inhabitants of some streets or courts are being decimated, those dwelling in others not far distant altogether escape; or, as frequently happens, the inmates of certain houses suffer severely, whilst their neighbors are entirely spared.

4. That the limits of a tainted district are sometimes clearly marked out. In illustration of this, my amiable teacher, the late Dr. W. P. Alison, Professor of Medicine of Edinburgh University, quoted a most striking example in his paper on "The Exciting Causes of Epidemics," in *The Medico-Chirurgical Review* for 1854. He wrote (on the authority of Ashton Bostock, Esq., Surgeon of the Guards), that *one* wing of a cavalry regiment, just arrived from England, and in high health, ascended the Ganges from Calcutta in boats, there being no cholera at the time in Calcutta. At a certain period of the voyage the troops arrived at a part of the country where cholera prevailed in the villages on the banks of the river, *but with which they did not communicate*. Here cases of cholera occurred in the boats; the men were advised to push on rapidly, and after a few days, when they had passed the limits of the existence of the disease on the banks, it ceased to show itself in the boats. What makes the case peculiarly conclusive is, that the other wing of the regiment followed afterwards by the same mode of conveyance, became "*affected with the disease at the same point, and lost it again at the same point.*"

Although very great differences of opinion prevail as to the part which obvious local causes of insalubrity bear in the production of cholera, yet it is almost universally considered that they are necessary for the development and propagation of this disease in its epidemic forms. Drs. Barton, Carpenter, Pettenkofer, and Snow, all agree in this general proposition. Pettenkofer, of Munich, maintains that a certain condition of soil is necessary to the local development of epidemic cholera. It must be pervious and permeable to water and air. It must possess a particular degree of *humectation*, depending upon the position of the subsoil water to the surface. It must be charged with organic, especially excrementitious matter. These he believes are essential conditions, *as to soil*, for the development of epidemic cholera. The nidus, or cholera poison, or cholera *ejecta*, once finding admission to such a soil,

undergoes those developmental changes which is characteristic of its rapid dissemination as an epidemic. Such a condition of soil fosters the multiplication of the cholera poison. It does not generate the poison *de novo*, but by means of the evacuations of those sick of the disease the fermenting contents of choleraic intestines find their way into such soils; and the aptitude for the multiplication of the poison by specific fermentation is in accordance with a certain state of *humectation* of the soil, which again has a definite relation to the recession of the subsoil water after it has approached unusually near the surface.

But apart from the local conditions as to soil insisted on by Pettenkofer, there is very strong evidence to show that the decomposing choleraic discharges (already in a state of active fermenting change in the intestine of the cholera patient) will produce the disease—which will go on changing and multiplying a virus capable of spreading through the air as well as through the medium of water. In the outbreak at Southampton in 1866 there were instances both of transmission by water and by air (Parkes, in Mr. Simon's *Ninth Report on Public Health*, p. 253). The discharges need not pass into the ground to decompose or ferment there. They can decompose equally well in sewers, and, of course, can propagate their specific fermentation to the contents of such sewers, and such contents may find their way as gases or more material elements in the air or in the water.

Impure water, lowness of sites, and the emanations arising from the decomposition of animal refuse, or of excreta, are the local causes now satisfactorily determined to have a more or less constant connection with the development and propagation of cholera.

That *impure water* has a powerful influence over the intensity of cholera outbreaks is now unquestionably established by the observations of Drs. Acland, Sutherland, William Budd, Parkes, and the late Dr. Snow, and the specific inquiries of the Registrar-General and Mr. Simon. Yet still it is found that *impure water* is not a necessary element in the generation of the cholera poison, as shown in the report of Dr. Baly (pages 201–205), Budd as to Bristol, Pettenkofer as to Munich, and Günther as to Saxony. From their evidence, “cholera can do its very worst where the drinking-water can play no possible part in its dissemination” (*Brit. Med. Jour.*, April 13, 1867, p. 416).

The localized attacks at Theydon Bois in 1865, in the east of London and in Southampton in 1866, all point unequivocally to impure water. With the *general outbreak* at Southampton, however, impure water had nothing to do. It had only to do with the production of the disease on board the steamship “Poonah” just before her arrival from Gibraltar, where she took in a tankful of very foul water, which, from its peculiar smell, evidently contained sewage (PARKES).

As regards London, it has been shown by Dr. Farr that *the elevation of the soil* has a more constant relation to the mortality from cholera than any other known element, the mortality from cholera being in the inverse ratio of the elevation. Yet, like the condition

of the water, the elevation of the soil has not been always found to be a necessary localizing condition; and there is now only left to be noticed *the influence of an atmosphere contaminated by the effluvia arising from decaying animal matter and excreta*. Dr. Cullen long ago remarked, and every industrious dissector knows, that the effluvia from very putrid animal substances readily produces diarrhœa. Yet it appears that the nature of the decomposing matter, and of the transforming process it undergoes, have some influence in modifying the effects on the human constitution. Districts in which the most putrid odors tainted the air have sometimes almost entirely escaped, whilst others contiguous to them have suffered severely. Dr. Chisholm (quoted by Dr. Alison in his paper already referred to) gives numerous pointed illustrations of this, in the cases of "bone manufactories," "manufactories for the conversion of dead animal matter into a substance resembling spermaceti," "of places where blood is putrefying, waiting to be used by sugar refiners," and "of leather-dressing establishments." In dissecting-rooms, where the process of animal putrefaction goes on to a great extent, diarrhœa is comparatively rare, if the rooms are kept clean. During my experience, as Demonstrator of Anatomy in the University of Glasgow, for a period of six years (including the severe epidemic of cholera there in 1848-49, and during which time almost all the subjects for dissection had died of cholera), not a single student suffered from cholera; and when the proper agents are used, such as the injection of arsenical solutions into the dead body, which have the effect of arresting and modifying the putrefactive changes, I believe the production of diarrhœa is an exception, and may be found to have as significant a cause in errors of diet or of drink as in too close an attendance in the dissecting-room. That the poison of cholera does not attach itself to the *dead body* (in a certain state of decomposition at least) is a fact confirmed by the experience of those connected with the dissecting-rooms in Edinburgh. It is certain that these were supplied during the greater part of 1848-49, as they were in 1832, almost exclusively by cholera subjects, and in neither year was there a single case of the disease among the numerous students attending these rooms (Dr. ALISON).

Much pains has been taken by Dr. E. H. Greenhow to investigate the precise conditions which, from their more uniform coexistence with cholera, might be supposed to produce or to aggravate epidemics of it. The result of his observations tends to confirm what Mr. Orton (*London Med. Gazette*, vol. x, p. 222, 1832) was the first of English writers to show—namely, that "an atmosphere impregnated with the products of *fermenting excrement* is at once the most obvious and most constant concomitant of cholera (*the privy or fecal contamination theory*). Such exhalations were often found, even in a concentrated form, in houses where the existence of any palpable cause of insalubrity would scarcely be suspected, and thus the fact is in some measure explicable, that the pestilence, sometimes passing over slums and rookeries, knocked at the door of the comfortable annuitant or the wealthy tradesman. It was found that persons appeared to suffer in proportion to the contamination of the air they

breathed with the 'privy odor,' and that immunity from this appeared to secure immunity from cholera." The observations of Mr. Orton and of Dr. Greenhow are confirmed by the investigations of Dr. Pettenkofer at Munich and at the village of Gaimersheim. Dr. Barton, of New Orleans, Dr. Milroy in his report on the epidemic at Kingston, Dr. Buckler in his account of the outbreak in the Baltimore almshouses, and Dr. Parkes at Southampton, give similar evidence confirmatory of the injurious influence of the *fermentive decomposition of animal excrement*.

The outbreaks of cholera in some of the camps in Bulgaria and the Crimea, especially at Aladyn and Alma during the war, also furnish sufficient illustrations; and I believe the outbreak of cholera at Scutari, in November, 1855, which suddenly commenced in the camp of the Osmanli Horse Artillery, had a similar origin.

Propagation of Cholera by Human Intercourse.—When cholera appeared in its epidemic form in this country in 1831, the majority of European practitioners were decided contagionists. Subsequently to that period a reaction of opinion occurred, and the question was discussed for many years without any definite result. In 1848, when the disease again became epidemic, many of the higher authorities coincided with "the solemn declaration of the Board of Health, that the malady was not in any way contagious, and that no danger was incurred by attendance on the sick." "A large body of evidence, however, now renders it certain that human intercourse has at least a share in the propagation of the disease, and under some circumstances it is the most important, if not the sole, means of effecting its diffusion" (Dr. BALY). Very positive evidence has now accumulated in abundance to prove the transmission of the disease by human intercourse. Healthy men carry the disease with them by their clothes, by their ships, and by their caravans. That such is the case, we have now ample evidence in the *Bengal Report*, of 1824, by Dr. Jameson; in cases related by Mr. Orton, in 1832, and by Dr. J. Y. Simpson, in 1838; in *The Edinburgh Monthly Journal* for 1849, by the late Dr. Cruickshank, at Dalmellington, in Ayrshire; by Dr. William Robertson, detailed in *The Edinburgh Monthly Journal* for August of that year; and more recently the account of the outbreak at Arbroath, in Scotland, in 1853, by Dr. T. Trail; and cases by Dr. Alison, in 1854, in the paper already noticed; in the report of Dr. Berg, of Stockholm, in 1848; in the Norwegian Reports of 1850-53; in the Report of the College of Physicians of London, in 1854, and the several reports of 1865 and 1866. These records afford undoubted instances which show that human intercourse is occasionally influential, in some way, in transmitting cholera into detached localities, where it may seize upon two or more individuals, and then cease. But it is no less certain that its general extension over the world cannot be accounted for by human intercourse alone, to the exclusion of aerial contamination. It is curious that in India, the birthplace and headquarters of the disease, the doctrine of contagion is almost universally disbelieved in by our professional brethren. The opinion generally entertained in India is opposed to the doctrine of contagion (Morehead, *Indian Annals of Med. Science*,

vol. i, p. 456). Such difference of opinion may admit of explanation in the fact that in India all the causes of cholera in its original home are in constant operation, and more especially prolonged heat, decomposing organic matters, a more or less debilitated state of the European constitution; and hence, no sooner is the poison or germ of cholera imported than the disease spreads with such rapidity as to resemble an epidemic invasion. Cases, however, of undoubted contagion are not wanting in India. Mr. Barry, of the Bengal service, records an outbreak of cholera at Gowalparrah, in Upper Assam, in 1853. In this instance cholera was evidently imported into a healthy place by a body of Sepoys coming from an infected locality. Every case of the disease could be traced to communication with the sick: a large number of attendants on the sick were seized, but those who separated themselves escaped in every instance (*Ind. Annals*, vol. i, p. 448. See also the instance of Dominica, twenty-two miles from Guadeloupe, referred to by Mr. Simon in the *Ninth Report of Public Health*, pp. 25 and 26. Also those referred to in the *Army Medical Departmental Report* for 1865, p. 349, given by Dr. Parkes from Dr. Günther's work on the Cholera in Saxony).

According to the accurate observations of C. T. Kierulf in the vicinity of Bergen, it appears that, when the disease is propagated by human intercourse, from one to four days elapsed from the supposed period of infection to the outbreak of the disease. Most frequently the disease appeared on the second day after exposure to the infection; and he found that the diarrhoea, so frequent during the invasion of cholera, is a part of the disease, and itself capable of infecting others with true cholera. This, Mr. Orton also showed, was the case in 1832 (*l. c.*) The extreme shortness of the period of incubation is an important element to be remembered in all investigations regarding the course of events in cholera epidemics. According to Dr. Budd's observations it seldom exceeds three days; and where the disease is virulent, there is evidence to show that it may not exceed six hours.

The inoculation experiments of Namias with needles loaded with the evacuations from cholera, and the experiments by tasting the vomited fluids by M. Foy and his coadjutors, have given entirely negative results. So also the influence of exhalations from the blood and evacuations of patients with cholera, as designedly experimented on by inoculation, has been of a negative kind. On the other hand, Lauder Lindsay, Marshall, Thiersch, and Meyer, have succeeded in communicating cholera to dogs and cats and mice, chiefly through the *rice-water* evacuations from cholera patients being swallowed by these animals.

Dr. Wm. Budd, of Bristol, maintains, with most cogent reasoning and evidence, that the poison is cast off by the intestine of the cholera patient in the characteristic rice-water discharges, and that it may be transmitted to other and uninfected persons in the following principal ways:

1. By the soiled hands of attendants on the sick; a mode of communication probably very common within the limits of the family circle.

2. By means of bed and body linen, and other articles tainted with the rice-water discharges.

3. Through the medium of the soil. The discharges being liquid, the great bulk of them find their way to the ground, from which the poison may be propagated in three ways,—(a) By rising into the air as a product of evaporation; (b) By percolating into the drinking-water; (c) By atmospheric dispersion in the form of impalpable dust, after it has passed into the dried state. It is, of course, difficult to establish these modes of propagation by direct proof; but circumstantial evidence, and evidence by analogy, is so cogent and weighty, that no reasonable doubts can now be entertained regarding these modes of propagation. By *experiment* the *enthetically* contagious poisons (*e.g.*, vaccine variola, woorara, &c.) are known to retain their properties in a dormant state for indefinite periods of time after having been dried up, and to recover these properties again when moistened. Evidence almost as certain as experiment demonstrates the same regarding the poison of scarlet fever, malignant pustule, glanders, and syphilis. Therefore, it is probably true of cholera, and the more so that the numerous and well-authenticated instances of the propagation of the disease through articles of dress shows that the poison, during its transit, must necessarily have been in a dried condition—a condition which entirely protects organic bodies from certain molecular changes; so that, so long as the material-holding poison remained in this dry state, no definite limit could be stated as to how long the morbid agent might retain its specific powers. From this point of view a single case may give rise to a wide-spread infection; and as cases multiply, it becomes more and more impossible to trace their lineal succession.

The relative share which the modes of propagation (here indicated by Dr. Budd) take in the propagation of cholera must vary with season and climate, with temperature, with the habits of the people, with the nature of the soil, with the water-supply, with the prevailing wind, and with general sanitary arrangements.

The experience of 1866 and of 1865 confirms all previous experience as to the propagation of cholera, so well summed up by Mr. Simon in his official memorandum of July, 1866. In it he assured the public that cholera is so little contagious, in the sense in which small-pox and typhus fever are commonly called contagious, that, if proper precautions are taken where it is present, there is scarcely any risk that the disease will spread to persons who nurse and otherwise closely attend upon the sick. But he admits it is not less true, that all matters which the patient discharges from the stomach and bowels are infective; that the patient's power of infecting other persons are due entirely, or almost entirely, to these discharges; that these, however, are comparatively non-effective when first discharged, but afterwards, while undergoing decomposition, acquire their maximum of infective power; that, if cast away without previous disinfection, they impart their own infective quality to other excremental matters; that if they get access, even in the smallest quantity, to wells or other sources of drinking-water, they may

infect very large volumes of water; but the infective influence of choleraic discharges attaches to whatever bedding, clothing, towels, and like things have been imbued with them; and that thus even a single case of cholera may exert a terrible power over large masses of population, *if local circumstances co-operate*.

The rapidity with which the rice-water discharges must pass into a dry state under the burning rays of a tropical sun, renders it highly probable, as Dr. Budd suggests, that in India "dust," bearing the poison of cholera in a dry state ("cholera dust"), has a large share in the mode of propagation; and when the disease has prevailed, the poison may be left behind in a dormant state, from being simply dry, so that seeds of a new outbreak may exist in the soil coextensive with the first. Hence the imprudence of encamping on old encamping grounds (to which I have so often referred); and, in short, these views of Dr. Budd appear to explain, in the most natural way, almost all the leading facts which characterize the diffusion of the pestilence. They explain especially the relation of cholera to filthy habits and defective drainage—its predominance in low levels—its striking tendency to follow the natural line of water-shed—its communication to persons who not only have never been in the presence of the sick, but who are stationed at a distance from them—contamination of those only who visited one particular or single privy, into which the rice-water evacuations had been discharged from the first casual case—and the operation of tainted privies in propagating the disease in workhouses, barracks, prisons, and places of public resort (Dr. Budd's Letters addressed to the *Association Med. Journal* in 1854-55; Dr. L. Lindsay's able papers; and Dr. Alison's paper "On the Communication of Cholera by Dejections," in *Edin. Med. Journal*, 1855; also Mr. Simon's *Ninth Report*, for 1866). Under circumstances, therefore, of great concentration, or otherwise, some unknown poison is communicated, probably by *fomites*, through human intercourse; and as emanations, of some kind or other, passing through the air, they act as poisons on the gastro-pulmonary mucous membrane of susceptible persons. Dr. Parkes has shown, in his Indian experience of cholera, that it may pass with extreme slowness even against the wind (and even the trade or monsoon wind), which only retards its course, but that a favorable wind promotes its transmission; and that it sometimes travels in this way, and not by the shortest route of human intercourse, or even by the route of greatest intercourse between places.

The communication of cholera by the so-called premonitory diarrhœa (*i. e.*, the early stage of cholera) is now beyond dispute. "An instance in point occurred at Southampton in 1866, where no cholera prevailed at the time. A man in the diarrhœal stage of the disease landed at Southampton, and went to his house, a clean airy place, where his wife and young child lived. He was laboring under great diarrhœa when he landed on Monday. On Wednesday following, his child was attacked with cholera, and died on Thursday; and on Thursday the man became worse, and died of cholera on Friday." Here the observations of Parkes confirm the observations of C. T. Kieruff, near Bergen, and of Mr. Orton, in 1832, already referred to.

[Cholera prevailed extensively in the United States army during the year 1866. There were 2813 cases, and 1269 deaths. It appears from Dr. J. J. Woodward's report to the Surgeon-General (*Circular No. 5, War Department, S. G. O.*, 1867), that it spread over the country during that year, extending as far westward as Forts Leavenworth, Riley, and Gibson, and in the southwest to Texas. In its progress it followed the lines of travel rather than any general westward course, and, in the case of the army, it especially followed the movements of bodies of recruits. The epidemic, so far as the army is concerned, evidently radiated from two chief centres—New York city, and Newport Barracks, Kentucky. The first reported case in the army, in 1866, was at Fort Columbus, Governor's Island, New York Harbor, in a recruit from the rendezvous at Minneapolis, Minnesota, of whose previous history and exposure nothing was known; he had been but three days at the post. In about an hour after his admission into the hospital another case occurred, also a recruit, with previous history unknown. Cholera was at the time prevailing in New York city. The fort too was in the immediate neighborhood of a quarter of the town chiefly infected, through which recruits passed with more or less delay. There was besides frequent daily communication between the fort and the town. Recruits from Governor's Island carried cholera to Hart's Island, another recruiting depot on the East River. The infection spread by readily traceable steps to Georgia; to Louisiana, by way of New Orleans; to Texas, by way of Galveston; to Louisville, Kentucky; to Richmond, Virginia; and to La Virgin, Nicaragua Bay. From Richmond it was carried to Norfolk, Virginia; from Louisville to Bowling Green, Kentucky. The probabilities appear to be that the disease was carried from New Orleans up the Mississippi River to various points on that stream, and west of it; and though the whole chain of evidence is not complete, yet there is a sufficient number of known cases of the transfer of the epidemic from one post to another in this region, to put this view of the whole movement beyond reasonable doubt.

The other principal centre appears to have been Newport Barracks, Kentucky, where the disease was plainly introduced from the infected city of Cincinnati, on the opposite side of the Ohio River. Although it did not prevail to any great extent at this post, yet it is in evidence that it was carried thence to Augusta and Atlanta, Georgia, and to Nashville and Memphis, Tennessee (WOODWARD, *l. c.*)

The following instances of the portability of the disease are cited from the official report.

1. On the 14th of July the steamship *San Salvador* left New York with seventy or eighty cabin passengers, and sixty in the crew and steerage. She touched at Governor's Island and took on board 476 recruits for the Seventh United States infantry. The men were lodged between decks, and were greatly overcrowded. On the second day out cholera appeared among the recruits, and when the vessel arrived at quarantine, near Savannah, Georgia, three deaths had occurred, and there were twenty-five ill of the disease. The troops were landed on Tybee Island. Cholera continued to prevail on the island during July and the first few days of August. Altogether there were 202 cases, and 116 deaths. The cabin passengers and crew of the *San Salvador* appear to have escaped, but of the ten white citizens residing on Tybee Island, nine were seized with cholera shortly after the arrival of the infected ship and five died. The tenth fled from the island, and is reported to have died of cholera some-

where in the interior of Georgia. No cases of cholera occurred among the troops stationed in Savannah.

2. The steamship Texas, with recruits from Hart's Island, left New Orleans, July 19th, and arrived at Galveston, Texas, on the 22d. The day after arrival one of the recruits was attacked with cholera, and died in thirty-six hours. In the outbreak which followed, 44 cases and 24 deaths are reported among the white troops at Galveston, and one fatal case of a colored soldier in the post hospital during August.

The portability of cholera was again amply shown in the army experience of 1867, by the movement of infected troops and trains. By these means the disease was carried, in July, from the Mississippi Valley, where it prevailed, across the Plains to every post on the Arkansas River and the Smoky Hill Ford. Three other notable instances of transplantation happened: one on the route between Forts Gibson and Arbuckle, one in the case of the posts in New York Harbor, and the third in that of certain recruits distributed from New York, by way of New Orleans, through Texas.

The importation of cholera into the posts of New York Harbor, in the summer of 1867, by an infected person, and the communicability of the disease, are clearly made out. The first case at Fort Columbus was on the 21st August, in the recruit Vassar, who had arrived at the post on the previous evening with a detachment of recruits from St. Louis, Mo., where cholera was prevailing. One man had died on the way with cholera symptoms, and Vassar had nursed him. On the 24th August, 144 recruits were sent from Governor's Island to Bedloe's Island, amongst them a man named Harden, who, with Vassar, had nursed the recruit who had died *in transitu*. Harden was attacked with cholera on the 25th August, and died. Subsequently during August and September there were 35 cases and 18 deaths at Fort Columbus, and 10 cases and 4 deaths at Fort Wood (*Circular No. 1, War Department, S. G. O., June, 1868.*)

Among the many striking and incontrovertible instances that cholera poison may be carried from one place to another by individuals, or by their luggage, is the outbreak of the Marseilles epidemic in the autumn of 1865.

Dr. Woodward, U. S. A., the author of the admirable and instructive Army Reports referred to and quoted from, remarks: "In a general way it may be said that the experience of the army in 1867 confirms the views in favor of quarantine formed during 1866, and especially confirms the opinions formed with regard to the danger of distributing recruits or other bodies of troops from an infected point to other garrisons (*l. c.*, p. vi). Early in 1867 the Surgeon-General had instructed medical officers, to endeavor, as far as possible, to protect any threatened command by a proper quarantine. The measures thus adopted, in conjunction with the hygienic precautions directed, undoubtedly saved many lives in the army, for the total number of deaths from cholera during 1867 was 230, and it cannot be claimed that the disease in itself was less virulent during 1867, for the proportion of deaths to the total number of cases was 1 death to 2.19 cases, while during 1866 it was 1 to 2.22" (WOODWARD, *l. c.*)*

* "At several points, as, for example, Augusta and Atlanta, Georgia, the epidemic did not extend beyond the infected recruits by whom it was imported. In many cases, however, it involved the rest of the command, and it is highly probable that this would have been the case far more generally but for the stringent hygienic precautions adopted.

"As a particular example of the value of such precautions, attention may be appropriately drawn to the appended extracts from the reports of Brevet Major E. Mc-

Those who argue against the availability of quarantine as positively protective against the dissemination of cholera, assert that it has never succeeded, and never can. The difficulty lies in making the means absolute. In proportion to its strictness is the risk from infected sources lessened. It is relatively, if not positively protective, and this view is fast gaining ground, as the result of a large body of facts. If efficient, it stops one mode of diffusion, and that a pretty potent one.

But admitting all that is claimed and probable of the dissemination of malignant cholera by human intercourse—body emanations, gastro-intestinal discharges, infected clothing—it is equally certain that, to account satisfactorily for its spread at times, we are obliged to own the agency of the atmosphere as a carrier, or an epidemic constitution, as well as the influence of localized conditions determining outbreaks and intensity. During the first two months after the French army landed in the Crimea, it lost more men by deaths and invaliding from cholera, than from gunshot wounds, from the battle of the Alma to the fall of Sebastopol; yet it brought no cholera with it, nor was there any at the time of disembarkation. The outbreak seemed to be due solely to atmospheric causes and insanitary conditions.

The organic theory as a cause of epidemics, first broached by Kircher, sanctioned by Linnæus, and ably advocated in late years by Sir Henry Holland, Henle, Dr. J. C. Nott, and others, was applied to cholera in Great Britain in 1849 and 1854, but, unsupported by observation, made but little headway. Recently there seems to be a tendency to a reconsideration of this hypothesis, which is certainly a very attractive one, and which would, if demonstrated by physical evidence, offer an easy and satisfactory solution of many of the mooted points surrounding the dissemination of cholera. "Many of the phenomena observed during the march of cholera epidemics," writes Mr. Goodeve, "might be explained much more satisfactorily upon the supposition of the exciting cause being masses of organisms moving in obedience to atmospheric impulses and currents, than by most other theories. They might multiply wherever they found a fitting nidus, which might be in privy atmospheres, or in air abounding in emanations from decaying and putrefying matter, or in crowded rooms, and, indeed, in all vitiated atmospheres. They might appear to impart an infecting character to the choleraic discharges by multiplying enormously in them" (Reynold's *System of Medicine*, vol. i, p. 147). Dr. Henry Hartshorne (*Cholera*, &c., 1866) is a decided advocate of the organic theory, and he believes that the cause of cholera is a (yet undiscovered) protozoon, or primal organism, of extreme individual minuteness, which, on entering the human body, affects it as an organic poison; and that the conditions which favor and maintain in life multiplication and migration, this *ens primalis*, are afforded by animal matter in a state of rapid and foul decomposition, along with moderately high temperature and ordinary moisture.

Dr. Lionel S. Beale, for some time engaged in the study of the poison of contagious diseases, has, as the result of minute microscopical inquiry, reached these conclusions: (1.) The contagious or infecting principle consists neither of insects, of animalcula, nor any kind of vegetable organism. (2.) But of living matter formed in the organism of man or

Clellan, Assistant Surgeon United States Army, from which it appears that cholera broke out at various points in the vicinity of Fort Delaware, in fact, encircling the post, but did not invade the garrison, although one case, which recovered, occurred in the family of an officer on the island." (*Circular No. 5, 1867.*)

animals—the particles being exceedingly minute and capable of retaining their vitality for a long time, and under various conditions, although separated from the body. (3.) That these living particles bear somewhat the same relation to the germinal matter of normal cells that pus-corpuscles or cancer-cells do. (4.) And that the living contagious particle is not, therefore, of the species of a *parasite*, nor can it be regarded zoologically as a species, nor has it originated in the external world, and grafted itself upon man; but it has originated in his organism, and is degraded living matter, descended from what was once normal living matter of the body itself.

Direct observation is yet lacking to the support of the organic theory, and however inviting and satisfying it may be, positive proof of the existence of the organisms will be required before its advocates can ask for its final acceptance. Future microscopical investigations of air, after the manner of Pasteur, may possibly settle the question.

In treating of the causation of cholera, Mr. Goodeve pertinently asks, whether it be not “more in accordance with facts to suppose that neither a miasm from without nor a miasm from within exclusively contains the specific poison? Might it not be that two factors are needed, the one some air-borne material or some dynamic modification of atmospheric elements coming from without, the other some local element, neither being potent unless united?” (*loc. cit.*.)

Symptoms and Various Forms of Cholera.—*Cholera Indica* has many degrees of severity, and hence many pathologists have divided it into *Cholera Indica mitior* and into *Cholera Indica gravior*. The French have termed the slighter forms of the disease *Cholerine*, and this name has been also recently used by Dr. Farr to designate the specific poison, or zymotic matter of cholera.

Malignant Cholera is divided into two stages,—the cold, pulseless, or asphyxiated stage, and the febrile stage, when the patient outlives the first. This latter stage, however, is not essential to the disease, and has been observed in India in a small proportion of the cases only. In Europe, however, the febrile paroxysm has followed in the majority of instances. The duration of the cold stage varies from a few minutes to twelve, twenty-four, forty-eight, or even more hours; while the febrile stage lasts from four to eight or more days,—making the total duration to vary from a few hours to two, three, or even four weeks.

The attack of this fatal epidemic is most commonly sudden, the patient at the time of his sickness being apparently in his best health; yet not unfrequently slight diarrhoea or other general indisposition has preceded it. In India, in some cases, the premonitory symptoms are vertigo, noise in the ears—the latter sometimes so loud as to have been compared to the humming of a swarm of bees, to the beating of drums in the camp, or the roaring of the surf on the Coromandel coast.

A classification may be made of the disease into three principal varieties, which coincide in their phenomena with many of the changes known to take place in the blood (PARKES).

1. The slighter forms commence with much watery purging and vomiting, and pass into the second and third varieties in varying

times. There may be from ten to fifty copious watery stools, and frequent copious vomiting, before there is any great loss of heat and failure of circulation. When purging commences twenty-four hours, or two or three or four days, before the violent symptoms, such as vomiting, purging, or cramps, such patients are said to have "premonitory diarrhœa." But there is always some degree of loss of heat and failure of circulation even in the slightest cases, else the case would be mere watery diarrhœa, attended only by exhaustion, and not by the symptoms peculiar to cholera. Cramps are seldom present till the stools put on the true choleraic character—viz., of copious white flocculi suspended in a watery fluid. The algide symptoms come on gradually, and are less intense than in the following forms; recovery is also more common. In the recent epidemic in London (1866), Dr. Sutton gives forty-one examples in which there was undoubted premonitory diarrhœa, the duration of the diarrhœa being as follows: In three cases, 12 hours; in one case, 18 hours; in one case, 19 hours. In seven cases, 1 day; in one case, 1 day and 9 hours; in twelve cases, 2 days; in six cases, 3 days; in in two cases 4 days; in two cases, 5 days; in one case, 6 days; in one case, 7 days. In two cases, 2 weeks; in one case, 5 weeks; and in one case, 8 weeks. In more than half the number of cases the diarrhœa preceded the marked symptoms by one, two, or three days; and of the forty-one cases, the diarrhœa in twenty-six was limited to the first three days.

2. If the poison acts with greater intensity, we have the second variety, in which there is less physical alteration in the fibrine, and the circulation is carried on for a longer time. Consequently, the characteristic change is not evidenced solely or chiefly in the interior of the vessels; but is partly transferred to the exterior of the vascular system. The albuminoid constituents, fibrine, and perhaps albumen, are effused in large quantities, and in all parts of the body, though chiefly on the free surfaces of the skin, alimentary mucous membrane, and more rarely the bronchial mucous membrane. The general nature of this effusion forms two characteristic distinctions between cholera and diarrhœa; for diarrhœa is a disease confined, in the first instance, to the eliminating part—viz., the large or small intestines, as the case may be—and is unattended, as a general rule, by the effusion of albumen and fibrine. The worst forms of this variety are seen in those cases in which, after two or three choleraic stools, severe and long-continued cramps come on, accompanied and followed by intense algide symptoms: after death the small intestines are generally found distended with the thick, white, flaky substance. Other cases of this variety present infinite modifications in severity, according as watery elimination is added to effusion of the fibrine; in other words, according as they tend towards the slighter forms.

3. Thus, if the final change at once occur, and there is a complete and rapid arrest of the circulation, either from the intensity of the cause or from constitutional predisposition, the worst variety is produced, in which "a mortal coldness comes on from the beginning." As the circulation is soon almost entirely arrested by physical alter-

ations in the blood—presumably, changes in the fibrine—there can be little purging and comparatively little sweating; there is always some effusion of the thick white substance into the intestines, but often little of the watery part of the blood. The symptoms might be inferred from a statement of this condition; we might have presupposed a very rapid loss of animal heat, loss of voice, deafness, and vertigo, total arrest of all secretions, defective aeration of the blood, consequent dark color of the surface, and early and deep coma.

The more usual course of the disease in this country, when limited to the cold stage, is as follows:

After the patient has been troubled for a few days with diarrhœa (the more insidious and dangerous because it is *painless*), but more commonly while he is yet in perfect health, and has retired to rest, and has slept soundly till the middle of the night, or even till early morning, he is suddenly seized with an unaccountable sickness and vomiting, together with a most profuse discharge from the bowels. More persons were seized from twelve o'clock at midnight to three o'clock in the morning than at any other time (Dr. Sutton, *Health Report*, p. 371, for 1866). These profuse evacuations are attended with severe pains down the thighs, and more especially by an indescribable and subduing sense of exhaustion, the patient often fainting in the water-closet. In an instant the physical powers of the body are not only exhausted, but its temperature sinks rapidly below the natural standard. An icy coldness benumbs it; while the skin is sometimes rendered so insensible that it has been known to resist even the action of boiling water or other powerful chemical agents. The breath, as it issues from the mouth, communicates a glacial sensation to the back of the hand; still, notwithstanding this great loss of temperature, the patient complains of being oppressed, and is incessantly throwing off the bed-clothes; while cold water, copiously and eagerly drank, is grateful to him; and, although it does not seem to afford relief to his insatiable thirst, it ought not to be withheld.

The extreme coldness of the first stage is further accompanied by a blue, livid, or purple discoloration of the hands and feet, extending not only a considerable way up the arms and legs, but sometimes over a great part of the body. These parts often become in a few minutes after the seizure, not merely shrunken, but singularly wrinkled, like the hand of a washerwoman after a day's hard labor. These symptoms are rendered still more distressing by the shrieks and groans of the poor sufferer, often tortured by spasms, which affect the fingers, the toes, the arms, and the legs—spasms which clench the jaw, fix the walls of the abdomen in contact with the spine, or draw the trunk into singularly contorted forms. The patient thinks he obtains some relief by the use of friction, and his cries to his attendants are incessant to "rub hard."

As the disease proceeds, the countenance assumes a character peculiar to this great struggle, the "*facies cholericæ*," the eye being deeply sunken, red, and injected; while the aqueous humor, transuding its coats, leaves the cornea flat and depressed as in the dead body; a broad and livid band encircles the lower portion of the

orbit; every feature, moreover, is sharp and pinched, as after a long wasting disease; the complexion thick and muddy; the lips and tongue purple. All these great changes have been known to take place in a few minutes.

In addition to this sad state, the vomiting is constant, the purging most incessant, and the pulse, though often natural, sometimes rapid, yet in some cases it is not to be felt, even from the first moment of the attack, either in the large superficial arteries or at the wrist; the voice is strangely altered; its firm and manly tone changes to a low, feeble, and unnatural sound. The urinary secretion is likewise entirely suppressed, while no bile flows into the intestines. The only organ which seems to preserve its powers is the brain; and the patient often to the last moment of his life retains the power of thinking, and of expressing his thoughts distinctly, sometimes full of hope, while at other times he seems indifferent to the fate which too often inevitably awaits him.

The symptoms characteristic of the collapse stage during the late epidemic (1866), according to Dr. Sutton, in the Cholera Hospital and in the London Hospital, corresponded with those witnessed in other epidemics. "The pulse was only just perceptible—that was with great care—or the patients were pulseless; the extremities were cold; the tongue was very cold, sodden, coated with thin white fur; marked lividity especially in adults; old people and infants were, as a rule, less livid; the voice was reduced almost to a whisper; the eyes were sunken, pupils dilated, conjunctiva white and glassy; hands sodden and shrivelled; the patient restless, turning from side to side, with the eyes for the most part wide open, or closed only for a few moments at a time; very wakeful; excessive thirst, to such a degree that little children would get out of their beds and go and place their mouths under the water tap; cramps in the calves of the legs, extending up to the thighs and walls of the abdomen, in exceptional cases into the upper extremities. The patients manifested a great indifference as to their condition. When the patients were in extreme collapse the purging often ceased, and that in some cases for some hours.

"In the worst cases of cholera the vomiting and purging began suddenly and violently, went on rapidly, the algide symptoms set in very early, and there was very little and often not any purging during collapse" (*Ninth Report on Public Health*, p. 381).

On the accession of the spasms, the vomiting, and the purging, the disorder is fully developed, and the crisis is at hand, which in a few hours must decide the fate of the patient. The termination may be favorable or unfavorable: if unfavorable, he may die with all the symptoms just narrated strongly marked; or, should it be favorable, they may abate, and a happier prognosis be formed. Unfortunately, however, it too often happens that, although the stomach retains what is taken, and the purging appears checked, and the patient falls into a doze, yet the weakness, the entire cessation of the pulse, the coldness and lividity of the surface, and the ghastly expression of the countenance, show that a few hours must close the scene, often with so little struggle that death is only marked

by the phenomena of cadaveric contraction, which sometimes continues active in the muscles for some hours after death. The largest number of deaths take place betwixt the hours of 7 and 11 A. M., and between 7 and 11 P. M., both with respect to males and females, (SUTTON, *l. c.*) These might be considered as the critical hours; and a true knowledge of such times would be of the utmost importance in the treatment, and especially the expectant treatment of the disease. The aim of expectant medicine is not simply to stand by and do nothing, but it is to watch the disease—to see if it is running its "natural" course—to judge whether the patient tends to do well; and if not, to ascertain how he tends to die, and to strive to counteract such tendency, and thus to gain time. It is very important, therefore, to know the hours when the vital powers are likely to be very feeble, and the vital functions almost brought to a stop: we may then assist the struggling patient at these particular times. "In any future registration of such facts," writes Mr. Simon, "it would be desirable to make separate enumeration of deaths in collapse, as distinguished from deaths in reaction and fever. Of course, too, in discussing the subject of hour of death, regard must be had to the hour of so-called 'attack'—*i. e.*, the hour of manifestation of severe symptoms; and if this should seem to be governed, at least locally, by some general law, the determining influence of local modes of life would need consideration."

If the patient should happily survive the cold stage, the disease may terminate by a rapid recovery, or it may pass into the second or febrile stage. The former is the more usual course in India, the latter in Europe. The first symptom of returning health is shown by the patient falling into a sleep of unusual soundness, during which the respiration becomes light and easy, the pulse freer, while a gentle warm perspiration bedews the whole body. This grateful pause in the disease appears to be the result of the returning powers of life, uninfluenced by medicine, for it often occurs where none has been given. After this balmy slumber the patient awakes refreshed, and often recovers so rapidly that, in the natives of India, it almost resembles a restoration after syncope. In all the presidencies, indeed, and especially in Bengal, the recovery of the European has, in general, been followed by a stage of reaction, usually slight, but in some cases assuming the form of the bilious remittent fever of the country, and which has occasionally terminated fatally.

In Europe, restoration after the cold stage, and without febrile reaction, is by no means so frequent or so rapid as in India. Sometimes the reaction is trifling, and sleep may indeed have ensued, fecal evacuations containing bile may have passed, the urine may again have flowed, the purging, vomiting and spasms may have subsided, the pulse may have risen, the blueness may have disappeared, and the temperature of the body may have increased, yet in many instances this amelioration of the symptoms has been only temporary;—the patients relapsed and died.

In most cases, however, the reaction was more considerable, and the patient, in a few hours after the subsidence of the cold stage, labored under a severe form of fever, in no degree dissimilar to, and

not less fatal than, typhoid fever. These *typhoid* symptoms, common in Europe and America, are said to be unknown, or nearly so, in India, where, if a secondary fever ensues, it assumes the form of the remittent fever of that country. But remittance is characteristic of typhoid fever; and this character may only be more expressed in India than in Europe. For the first few hours after the febrile reaction commences the tongue is white, but it quickly becomes brown and dry, while black sordes incrust the teeth and lips. The eye becomes deeply injected and red, the cheek pale or flushed, the pulse rapid, and the temperature of the body a little above the natural standard. The patient, either delirious or comatose, then lies in a state resembling the last stage of the severest typhoid fever of this country. This struggle usually lasts from four to eight days, when the symptoms either gradually yield or death ensues. In a few mild cases the fever assumes an intermittent type, or sometimes a quotidian, sometimes a tertian form: all these cases usually recover. Such is a general outline of the symptoms of this formidable disease.

The blood in cholera varies according to the stage of the disease. In the cold stage it is usually of an unnaturally dark color and thick consistency, so that it flows with difficulty from the veins, and very imperfectly separates into clot and serum. Blood taken from the temporal artery has been found equally black and thick. After the secondary fever is formed, the quantity of serum increases, till at length it is much more abundant in the blood than natural; and it is singular that this takes place notwithstanding that the secretion of urine is re-established.

Duration of the Disease.—It is of importance to determine whether the cholera process is limited in its duration. Dr. Sutton has attempted the observation; and finds that while the “cold stage” is always present more or less, the “hot stage” may be absent, and is no essential part of the disease. It varies very much when present. In mild cases it is very short and scarcely appreciable. In severe cases it is long and protracted, at least in this country. The phenomena of collapse appear not to be limited to any definite time. Some patients became collapsed very early; others not until vomiting and purging had continued several hours, and was then often *not* protracted. In the milder cases the algide symptoms were scarcely, or even not at all, marked; and all experience has shown that the collapse of cholera is not always present.

Dr. Sutton then endeavored to ascertain what period elapsed from the time when a patient was seized with the characteristic symptoms of the cold stage—the violent symptoms—to the time of his entering reaction. He finds there are good reasons for believing that the *cholera process* runs a definite course of from twenty to thirty hours.

Relation of Vomiting and Purging to Algide Symptoms.—When patients went into collapse the vomiting and purging very greatly diminished, and in some cases entirely ceased; and some of the worst cases—cases which seemed almost sure to prove fatal—had very little, and often not any, purging. Thus cases characterized by the most continued purging and vomiting were not by any means

the worst class of cases—for the most part the very opposite. It was the exception for such cases to pass into collapse; and if so, the algide symptoms came on slowly.

It by no means follows, because a patient is very much purged and vomits violently and very frequently, that he is in a worse condition than another patient who vomits and is purged much less. Dr. Sutton brings forward evidence which clearly shows that the algide symptoms were not in regulated proportion to the frequency of the vomiting and purging. He shows that a patient may be purged hour after hour, may almost continually vomit, yet may not pass into collapse; whereas another patient is purged and has vomiting for two or three or five hours, and passes into deep collapse. If the collapse be dependent solely on the loss of fluid, it is difficult to understand why in the very class in which there is protracted purging there is the least collapse; and even on the assumption that one patient passes in two or three evacuations more water than another does in double the number, and admitting that it is so in some cases, we are yet called upon to explain how it is that a patient who is not purged at all—that is, has had no discharge from his bowels—dies very suddenly; that another who has only been purged four times passes into collapse and dies; while others are purged twenty or thirty times without ever showing any well-marked symptoms of collapse. It is difficult to conclude that one patient passes more fluid in one evacuation than another does in twenty; and any explanation of this difficulty, to be satisfactory, must take into consideration not only the quantity of fluid withdrawn from the blood, but the rapidity with which it is withdrawn.

In the worst forms of cholera a considerable quantity of water and other constituents of the blood are withdrawn very suddenly from the system; and there seems to be a decided relation between the severity of the collapse and the rapidity and violence with which the cholera process sets in and is carried on.

Evidences of Reaction.—When a patient dies in advanced reaction, or was in complete reaction at the time of death, the different organs of the body regain their weight, sometimes even weighing heavier than usual. This is especially the case with the lungs. In collapse the lungs and spleen weigh much lighter than normal.

In favorable cases of reaction the wakefulness characteristic of true collapse gives way gradually to sleep. The color returns, the pulse becomes distinct and more perceptible, and in some cases the patient may sleep quietly for some time.

The thermometer, in the experience of Dr. Sutton, is of all single guides the best, but cannot be absolutely relied on, unless the temperature in the axilla be very low indeed, and in the rectum very high: *e. g.*, if the temperature in the axilla be 92° Fahr., and in the rectum 102° Fahr., the patient is still in collapse; but if the temperature in the axilla were 95° or 96° , imperfect reaction may be commencing. Some parts of the body appear to pass into reaction before others; and imperfect reaction is sometimes associated with bloody evacuations; and if a patient is pulseless, but with a natural

color and a greasy perspiring skin and a coated tongue, he will in all probability pass bloody evacuations, and then will certainly die. Dr. Sutton thus recognizes a class of cases having the following symptoms: The patient is seen lying on his back, eyes open, looking very wakeful, mind collected, voice weaker than natural, at times the typical choleraic voice, color natural, lips natural, complexion greasy, tongue sometimes cold, livid, of gray color and covered with white fur; at other times the tongue is warm and coated with yellow fur. The hands are of a livid red color, cold, and shrivelled. The temperature in the axilla is generally lower than usual. Respiration is labored, and generally accelerated—often 25, sometimes 40, a minute. The pulse at the wrist may be only just perceptible, and very often such patients are pulseless; there may be no purging for hours together, and very little vomiting. There may be profuse perspiration, the face and hair wet with it. The patient may lie for hours like this, and even one or two days. Such bloody evacuations appear on an average about twenty-eight hours after the violent symptoms of cholera set in.

When the algide symptoms are most severe the reaction is greatest and most protracted. The longest reaction was seventeen days, the shortest sixty hours, in Dr. Sutton's experience. In the mild cases the longest reaction was seven days and the shortest twelve hours. The duration of suppression of urine is also in proportion to the severity of the algide symptoms. In one case no urine was passed, and none discovered in the bladder, for six days and ten hours; in two cases none for five days, and in two none for four days. In the milder class of cases three days was the longest period of suppression, and the shortest ten hours.

Chemical Changes undergone by the Body in the Progress of Cholera.

—Dr. J. L. W. Thudichum, at the instance of Mr. Simon, made important observations during the epidemic of 1866, which are published in Mr. Simon's report of that year. The following is a summary of his results:

The blood after death during collapse contains urea in variable abundance. The rice-water like evacuations contain butyric acid, and yield nitrogen and carbonic acid, but no urea. In bodies dead at an early period there was no urea; and more seemed to accumulate after a protracted algide stage, and much more after three to six days' torpid condition; and the greatest amount of urea was found after a long algide stage, with rise of temperature at the end.

The secretion of bile is completely arrested; and in extreme cases a clear white fluid percolates through the hepatic ducts, free from bile, coloring matter, and albumen. It seems to be simply water, with a trace of alkali and a vestige of mucus. In some instances the fluid is colored, but contains no bile acids. The bile ducts shed their epithelium.

The blood loses water, albumen, and salts, and is incapable of passing the capillaries with the usual freedom. It retains most of its coloring matter in its normal chemical composition; and Dr. Thudichum's observation led to the conclusion that any fermentation of

the blood was very improbable in the manner in which the intestinal contents are fermented. The blood absorbs water from the tissues; and there is no chemical evidence of any special cholera poison in the blood. The epithelium of the inner surface of the bloodvessels becomes detached and mixes with the blood, and the blood adheres to the bloodvessels with great pertinacity.

The Epithelium.—Death and desquamation of the epithelia pervade the entire surface where epithelium exists.* The *tissues* generally are doughy to the feel, from want of water. The muscles become dry and are affected with cramps; the spasms, beginning at the most distant parts, rise gradually to the centres; and the cramped muscles show deposits; urea accumulates in them, and nuclei of their sarcolemma multiply.

Intestinal Contents and Rice-water Evacuations.—The contents of the colon half an hour after their removal were observed to evolve gas, and to lift off the heavy glass stopper of the bottle in which they were contained. Their smell was putrid, but not fecal. The flocculent deposit which formed on standing filled one-half of the bulk of the fluid. It consisted of intestinal epithelium in patches, single cells, and cells in all conditions of disintegration, and great numbers of vibriones. The fluid filtered but very slowly, and the filtrate was not clear. Its reaction was strongly alkaline. On dialysis of the alkaline rice-water, the *dialysate* gave an acid reaction, from which, on being neutralized by baryta water, and the fluid evaporated, several matters could be obtained, namely,—(1.) A body crystalline like leucine, and combining with nitric acid; (2.) An oily substance, which was soluble in water, and with nitric acid gave a peculiar pink reaction; (3.) Butyric acid, combined with the added barium; (4.) Inorganic salts in considerable quantity; (5.) No urea could be discovered in the rice-water stools.

Volatile acids, butyric and acetic, were obtained also from the rice-water stools and intestinal contents, the latter apparently prevailing in quantity. They were combined with ammonia in the original fluid. The rice-water evacuations, therefore, contain the following ingredients: Vibriones, cells from the surface of the intestine, granular debris of cells, mucine, modified hemochrome, albumen, albuminous body giving rose-pink reaction, butyric acid, acetic acid, ammonia, leucine, inorganic salts. Nearly the same results

* In all of this description post-mortem results are unfortunately mixed up with vital phenomena; and it is to be regretted that evidence is so contradictory on a matter of fact so definite as the shedding of epithelium during life. So long as the patient lives and passes the characteristic evacuations, I have never seen them containing shed epithelium. It is only in the post-mortem contents of the bowel (small intestine) that cylindrical epithelium is to be found—the result, no doubt, of maceration. It is alike found in the stomach, urinary and gall bladder. Denudation of the intestinal villi, or desquamation of the epithelium, is no essential lesion of cholera. Boehm's statement seems to have been adopted as one of authority, without further question; and while competent observers have agreed with him (*e. g.*, Beale) on the one hand, no less competent observers have not been able to confirm it on the other (*e. g.*, Parkes, Gairdner, Lauder Lindsay). The observations at the London Hospital (*Report*, vol. iii) and those of Dr. Bruberger, of Berlin, who examined the stools of 640 cases, confirm the statements of Parkes, Gairdner, and Lindsay (*Virch. Arch.*, 1847, p. 261, quoted by Parkes in *Army Med. Report* for 1865).

were obtained by Dr. Lauder Lindsay in 1853. The evacuations are in an active state of decomposition, and evolve gas, which at first is composed almost entirely of nitrogen; soon, however, carbonic acid prevails, and ultimately nothing but carbonic acid is evolved. At one period some hydrogen is developed. In 1848 Dr. Parkes examined many cholera stools, and his observations coincided with those of O'Shaughnessy, Vogel, Wittstock, and Andrew Buchanan (of Glasgow). The thin fluid was always alkaline, and contained an abundance of alkaline chlorides, phosphates, and sulphates, and a certain proportion of albumen. The *odor* was always peculiar.

Dr. Thudichum cannot discover any specificity in the above ingredients; but many of them are analogous to the products of ordinary processes of putrefaction. If it is admitted that the cholera evacuations acquire infective powers only after a period of fermentation, it is also easy to understand that the specific infecting power may belong to albumen or mucine at a particular stage of disintegration or chemical cleavage. The next knowledge which it is necessary to acquire is evidently this—namely, the exact period at which the rice-water stools acquire infective properties, and their chemical composition at that period. The most dangerous period of the choleraic stools is believed to be when they become very ammoniacal. This occurs usually immediately they are passed, but not to any extent for some time; and anything which makes and keeps them acid prevents the ammoniacal change (PARKES).

Urine in Cholera Reaction.—As the complete suppression of the urinary secretion in collapse, lasting for hours or days, is one of the most striking and peculiar features of cholera, so its reappearance is amongst the earliest and most auspicious signs of beginning recovery. The first secretion mostly contains the evidence of the mechanical obstruction of the minute channels of the kidneys, and of the general death of the epithelia of the urinary passages. It also contains the sign of continued resistance to the blood-current through the kidneys in the form of transuded albumen of the blood. And in many cases it carries small quantities of peculiar abnormal ingredients, which may perhaps be products or remnants of processes engendered by the choleraic process in the blood. The quantity is at first very small—urea much diminished.

[*Temperature in Cholera.*—Though observations on this point were made in 1831, the first which merit attention are those of the epidemic in Europe of 1848–50. But the statements of observers differ widely, for whilst Von Bärensprung and others assert that there is in the algide state a general fall in the temperature of the body, that the loss of heat is not only external but general (Müller's *Archiv*, 1852), Zimmerman, from two observations, came to the opposite conclusion (*Deutsche Klinik*, 1856). Briquet and Mignot believed that the greatest fall of temperature took place in the algide state, though they admitted to having in a few cases found a rise. Similarly opposed statements were made by others. All these measurements, there is reason to suppose, were axillary, and, therefore, of little value; for in the algide state the temperature of the axilla is considerably lower than that of the natural outlets, and it is

impossible in this manner to ascertain the general body-heat. This can only be accurately determined by placing the thermometer in the rectum or vagina, which do not suffer from the rapid loss of heat. In the first stages of cholera the temperature of the closed axilla, compared with that of the vagina or rectum, shows a difference of one or more degrees. In the stage of reaction it is as trustworthy as in other disorders.

In the observations made at the Salpêtrière, Charcot found the temperature of the rectum raised in the algide stage in every case but one, some of the highest temperatures being met with at the same time that the coldness of surface and cyanosis were most marked, and where death soon followed. In case No. 2, 105.3° Fahrenheit were noted five hours before death, and one hour after death it had fallen to 104° (*Gazette Médicale*, 1866).

Messrs. J. McCarthy and Dove found the extreme ranges in the disease 91.2° and 105.6° Fahr. In the stage of reaction the temperature was often below the normal standard throughout, and was sometimes even lower than it had been during collapse. They remarked that in collapse there was a considerably higher temperature in the vagina or rectum—in one case a difference of 5.4° Fahr.; and that as the case progressed the internal temperature fell, while the external rose, till they were again equalized (*London Hospital Reports*, 1866).

The following are some of the conclusions reached by Mr. F. Mackenzie, as the result of careful observations made by him, in the London Hospital: (a) The temperature of the axilla will rise 2° Fahr. after a severe attack of cramps; this may be caused by muscular exertion and consequent acceleration of respiration at the same time. (b) The frequency of respiration and the temperature in the rectum or vagina seem to have some relation to each other; the more hurried the respiration the higher the temperature will be. (c) Cases in which the respirations are more than 40 in the minute, and the temperature above 101° Fahr., rarely recover. (d) The body-temperature in roseola cholera is raised in proportion to the severity of the eruption (*Lond. Hosp. Reports*, vol. iii).

Dr. L. Güterbock, physician to one of the Cholera Hospitals of Berlin, has given the results of a series of very extended and careful observations of the thermometry of cholera, the measurements being made in the vagina and rectum, in the following proposition:

(1.) In the algide stage of cholera there occurs a remarkable cooling of the parts of the body attached to the trunk (head, extremities), such as is found in hardly any other disease. (2.) In the algide stage of cholera, the temperature of the vagina and rectum is the highest (measurable) in any part of the body, and alone can determine the true temperature of the entire body. (3.) In the algide stage of most cases, whether they end in death or recovery, the internal temperature of the body is raised; occasionally normal; and very rarely lowered. The cause of this difference is not as yet apparent in the clinical history, or in the pathological appearances after death. (4.) In the algide stage, the temperature of the entire body usually rises with the approach of death, and up to its occurrence; and no elevation of it seems to happen after death; yet cases occur where at the death-struggle there is no rise, and with no explainable reasons. (5.) With the beginning of uncomplicated reaction no rise of temperature takes place, but more usually a slight cooling of the internal parts, whilst the surface becomes warmer. (6.) In cases of protracted reaction (prolonged asphyxia), the temperature of the whole body commonly sinks below the normal standard. (7.) In a great proportion of cases, during the inflammatory sequelæ, there is a decided rise of tem-

perature of the whole body. (8.) During complete convalescence an abnormally elevated temperature is frequently ascertained, without any apparent pathological cause (Virchow's *Archiv für Patholog. Anat. und Physiol.*, Jan., 1867; *Glasgow Med. Jour.* May, 1867.)]

The Temperature in Cholera, as determined by Dr. Thudichum, falls steadily from normal to 5.4° Fahr. or 7.2° Fahr. below it, and in most cases very rapidly.

The lowest temperature is quickly reached in deepest collapse; and the minimum temperature of all cases observed in the algide stage are below the lower limits of the fluctuation of health. The maximum temperature of the majority of cases observed are below the upper limits of the fluctuation of health.

The lower the temperature and the longer the duration of the algide stage, the higher and the longer continued is, on the whole, the temperature of the tepid stage, which does not exceed the upper normal, unless the temperature of the algide stage had previously sunk below 95° Fahr. But the temperature may for a short time reach 95° Fahr. or less, and yet the temperature of the tepid stage not rise above the upper normal.

When the maximum temperature of a case of cholera remains throughout below the normal average, the case will probably be fatal. Among the thirty-nine cases observed, all such cases, seven in number, proved fatal.

On the basis of the thermometric observations alone, cholera may be divided into two stages,—the first or algide stage, from the beginning of symptoms to that period where temperature reaches again the normal limits or average; and the second or tepid stage, in which temperature either remains within the normal limits or rises more or less above them, in some cases even to febrile height, afterwards descending again to normal limits.

But on the basis of all the pathological phenomena and clinical data, the following seven stages of cholera may be distinguished (THUDICHUM): (1.) *Fæcal diarrhæa*; (2.) *Choleraic diarrhæa and vomiting*, quick sinking of temperature; leading to (3.) *Asphyxia or collapse*, in which lowest temperature is reached; (4.) *Reaction*, which may be defined as the cessation of collapse and the beginning of the re-establishment of the suppressed functions; (5.) *Torpid stage*, or secondary period of algide stage, in which, reaction notwithstanding, temperature remains below the lower normal limits, and then gradually or suddenly rises to the normal average; (6.) *Tepid stage*, in which, during continued reaction, temperature rises to normal or its upper limits, more rarely somewhat above; (7.) *The febrile stage*, only reached in cases where the entire algide stage has been very long, or where there are complications, or secondary lesions arising out of the choleraic process. Reaction does not always terminate the algide stage. For although, from the moment of the beginning of reaction, temperature rises somewhat in most cases, in exquisite cases it does not reach the lower limits of normal fluctuation. The algide stage is evidently continued into the state of reaction, and the tepid stage is the result only of continued reaction.

Reaction begins mostly with absorption from the intestinal canal within thirty-six hours from collapse, possibly also with some actual secretions.

The influence of the temperature of the external air upon the temperature of the choleraic process was imperceptible during the observations recorded by Dr. Thudichum, as its range was very equable during the time of observation, the wards being mostly at 66.2° to 68° Fahr.

The lowest temperature observed in any case which recovered was 92.8° Fahr. It is at present uncertain whether there is a minimum temperature below which the body (axilla) cannot be cooled down without fatal results. If there is such a temperature it will probably be about 92.3° Fahr. *A low minimum temperature is at present of less significance than a low maximum.* All cases of cholera, the temperature of which ranges persistently below the lower healthy limits, even if no very low minimum temperature was reached, seem to have a fatal prognosis.

"It is all-important to remember," observes Mr. Simon, "that the thermometric observations recorded in this instructive section of Dr. Thudichum's report are exclusively of *external* temperature. In order to a complete understanding of the thermal phenomena of cholera, observations of this kind require to be supplemented by observations of *internal* temperature." And Mr. Simon therefore refers to some such, which have been elsewhere recorded.

In forty cases of collapse treated in the London Hospital in the late epidemic, temperatures were measured, simultaneously in the rectum or vagina and in the axilla, by Mr. F. M. Mackenzie, assistant resident medical officer of the hospital.* Another important set of double observations has been published in Germany by Dr. Güterbock. From these observations Mr. Simon concludes that "the choleraic affection of the bowels is a heat-making or inflammatory" process, on which the development of inflammatory fever, by circulation of blood from the inflamed parts, would as a matter of course attend, were it not that circumstances special to the disease (but accidental) suppress or circumscribe the manifestation. In a typical case of collapse the axillary thermometer shows a temperature perhaps little above 90° Fahr., while a thermometer in the rectum or vagina is marking a temperature high above the normal. With the superficial pulselessness of collapse before one, the suspicion cannot fail to arise that this vast difference of temperature between external and internal parts denotes mainly the failing blood-supply of the former; a state which, in so far as it does not equally affect all parts in the aortic circulation, may not improbably "be deemed to depend on the muscular contractility of peripheral arte-

* Mr. Mackenzie's notes of these observations are published in the third volume of the London Hospital Reports. Besides his observations, others, also made in the East London epidemic, are mentioned more or less fully in the same volume—viz., a few made in the London Hospital by Messrs. McCarthy and Dove, and some made, in the Wapping Cholera Hospital by Dr. Woodman and Mr. Heckford.

ries." It is not yet determined whether the general temperature of the blood is not febrile.

As regards temperature, the following conclusions were arrived at by Surgeon A. Leith Adams and Assistant-Surgeon F. H. Welch during the epidemic at Malta, of which they have given so admirable a report (*Army Med. Dep. Report*, vol. vi, 1864, p. 341):

"1. That a strongly marked, rapid downfall from the average normal temperature, 97° , takes place soon after the setting in of the cholera symptoms, and the extent in proportion to the dose of the poison; the downfall being characterized in the healthy young, and up to middle age, by elevations and depressions, each succeeding one of the latter reaching a lower point than the preceding one; in the aged, weak, or debauched constitution by an uninterrupted sinking of the thermometer. The average fall from normal temperature into collapse was 11° , the extremes 7° and 15° Fahr.

"2. That the highest temperature at which the general symptoms of collapse became apparent was 90° , the lowest 82° , the average 86° Fahr.

"3. That during the period of collapse the temperature underwent but slight variations in the aged, weak, or debauched; while in the young, and up to middle age, it was characterized by undulations.

"4. That the stage of complete collapse is not marked by any characteristic unvarying point of temperature. It would seem that an excessive dose of the poison is accompanied by a corresponding loss of heat; but when the vital stamina is deteriorated by drunken habits or delicacy of constitution, either the general symptoms of collapse are present when the thermometer makes no great fall, or an excessive lowness is reached with no corresponding general indications—*e. g.*, Cases 1, 2, 6, and 13 [in their Report]; while, on the other hand, a hardy well-used constitution does not betray signs of failing until the respiratory function is much interfered with. The lowest point reached during life was 73° .

"5. That a general brightening up of the patient, unaccompanied by any change of temperature, often preceded the final downfall, and was exceedingly deceptive until appreciated rightly.

"6. That the general signs of reaction were preceded by a marked elevation of the temperature, and when convalescence ensued, this reaction was characterized by fluctuations tending towards reinstatement of normal temperature. The average rise from complete collapse into full reaction was 6° , the extremes 8° and 4° .

"7. That when death ensued, whether preceded by reaction or not, the fall of the thermometer was most marked and rapid.

"8. That after death a rise of temperature ensued in the cases of great severity and quickness of course; but when the disease was prolonged, the patient falling into that senseless condition well expressed as 'death in life,' the contrary was the rule.

"9. The readings of the hands and epigastrium followed the breath's variations, though not always in the same ratio. As will be seen, the epigastrium especially showed a great tardiness in assimilating itself to the others, and was very tenacious of its heat.

"Thus, the thermometer indicated that in the aged and delicate the vital powers gave in to the poison, step by step, commensurate with the dose; collapse reached, a comparative quietness ensued, followed by the system asserting its superiority, or succumbing rapidly. The course from the onset to the termination was gradual, with no marked deviations.

Not so, however, with the young and healthy. Although the system was compelled to give way to the attacks of the virus, it was not without a struggle; the collapse was marked by constant attempts at reaction, and this having once set in, the vital powers seemed to overreach themselves in their eagerness to resume their normal condition.

"As helps to prognosis, it may be said that a rapid and marked fall at the onset, a temperature below 86° , a further loss of heat during collapse, a setting in of general symptoms of reaction not preceded by rise of temperature of breath and hands, each and individually indicated badly; but it must be borne in mind that the previous habits of life were great influencing causes, and this more especially when drunkenness was the deteriorating agent. This vice, *per se*, appeared to predispose to the disease; but no words can express its baneful effects as demonstrated by the manner in which its devotees succumbed to the poison, even when in a minor state of intensity."

Predisposing Causes of Cholera.—The influence of these is chiefly apparent in the age and sex, food, fatigue, filth, misery, and intemperance of the people.

Both sexes and all ages, including new-born children, are liable to the disease. Dr. Farr's results show that males suffered more than females at all ages under twenty-five years, but between twenty-five and forty-five the females suffered more than the males. The deaths from cholera in Paris were estimated at 18,402 in 1832; and it was remarked that the mortality was least from six years to twenty, greater from thirty to forty, and greatest of all in old age. The influence of *sex* in predisposing to cholera can hardly be said to be determined; for in Calcutta, of the native inhabitants attacked with cholera, the males were to the females as four to one, while in Bombay the proportion was as seven to twenty-five. In Canada the soldiers' wives were observed to suffer nearly in an equal proportion with their husbands; and this was the case among the civil inhabitants of Gibraltar.

In all countries the *lower classes* have always suffered in a much greater proportion than the upper classes. In Calcutta the disease ran a wide career of destruction in the native town, while the "City of Palaces," inhabited by the English, was much less affected in proportion to their numbers; and the same disproportion has been observed in Bombay. In general, it has been observed, among the native inhabitants of India, that the Brahmin and Banian merchant suffered less than the Ryot or farmer, while the poor outcast Pariah suffered the most of all. In every town in Europe it has been observed that the lower classes, and especially those resident on the banks of rivers, have suffered infinitely more than the upper classes.

In military life it has been believed that the Sepoy suffered more than the European soldier living in India. This, perhaps, is true in some instances; but the returns of the Madras army show this not to have been the fact in that Presidency. There the European soldiers attacked appear to have been as one to three, while of the Sepoy force it was only one in four and a half. In the Indian

army, also, it appears to have been universally observed that the officer suffered in a less proportion than the soldier, the cavalry than the infantry, and the infantry less than the hard-laboring ill-fed camp-follower.* The troops on march likewise universally suffered more than the troops in quarters; and this influence of long marches appears to indicate something more powerful than mere fatigue in bringing about the disease. Dr. Balfour has proved that of the native soldiers of the Madras army thirty-two died of cholera in cantonment, and eighty-six when marching, to an average of 10,000 strength; the number attacked being respectively 85 and 200 in 10,000. Dr. Lorimer's reports show that the men were more frequently attacked on long than on short marches, the men (as Dr. Farr observes) being longer exposed to the causes of disease. These causes are those which are incidental to the life of a soldier on the march, such as lying by the banks of rivers, on low marshes, jungly grounds, sleeping on the ground, and encamping amongst the filth of encampments recently occupied, but abandoned—of which indiscretion there were many melancholy examples during the war with Russia in 1854; for example, the occupation of the evacuated camping ground at Aladyn in Bulgaria, and that on the heights above Alma, previously occupied by the Russians, the consequences of which were so fatal to the first and fourth divisions of our army.

The effects of a poor diet in predisposing to cholera will perhaps be better understood by stating that the European suffers less than the Mohammedan, and the Mohammedan, who is better fed and better clothed, than the Hindoo, except during their rigid fasts, when the Mohammedans suffer in a much larger ratio. During the epidemic of 1848 and 1849, in Edinburgh, Dr. William Robertson of that city found that anæmic persons were those most predisposed to cholera.

Prognosis.—The mortality from cholera in all countries is very great. Taking the whole number attacked, it is said that the number of deaths in Astrakan were as one to three; in that of Mishni Novogorod as one to two; in Moscow and Kasan as three to five; and in Penza, in the country of the Don Cossacks, as two to three. In the summer of 1831 the mortality at Riga, St. Petersburg, Mitau, Limburg, and Brody, according to the *Berlin Gazette*, was about one-half, while at Dantzic, Elbing, and Posen, it was about two-thirds of the whole number attacked. The period of the epidemic, how-

* The Madras Sepoy, of whom alone Dr. Balfour wrote, invariably carries his family with him. At the end of a long march he puts off his accoutrements, and hastens back, without tasting food, to assist his family out of the difficulties incident to a country in which the roads are often mere tracks. He thus often performs nearly double the route march, and finally encamps on ground which for years has been used for the purpose, and is saturated with the excretion of former sufferers from the disease. Moreover, for a long time the authorities in Southern India were most reckless in sending regiment after regiment in one another's footsteps, through districts known to be infected; and as they all occupied the same encamping ground, the last regiments pitched in places saturated with cholera evacuations, and surrounded by the half-buried remains of the dead. These facts to some extent explain the effects of marching on Sepoys (W. C. MACLEAN).

ever, greatly influenced the mortality; for, on the first onset, nine-tenths of all those attacked perished, then seven-eighths; and the proportion of deaths forms a gradually decreasing series of five-sixths, three-fourths, one-half, one-third, till, towards the close, a large proportion of those attacked recovered. The uniformity of this law in every country affected with cholera, whether Europe, America, India, or China, is extremely remarkable.

The chances of recovery are much diminished in young children and in the aged; the age of greatest number of recoveries being from fifteen to twenty. The feeble in constitution, the anæmic, the sick, and the convalescent, were in all cases the surest victims of cholera. But, whatever the age of the patient, Gendrin states he lost every case which became pulseless.

Treatment.—There are few diseases for the cure of which so many different remedies and modes of treatment have been employed as in cholera, and unfortunately without our discovering an antidote to the poison. In Moscow, it is said that the mortality was not greater among those destitute of medical aid than among those who had every care and attention shown them. It may be fairly inferred, therefore, that in the severer forms of the disease the action of this poison is so potent as to render the constitution insensible to the influence of our most powerful remedial agents. When, however, the disease is mild, or on the decline, much may be done, by obviating symptoms, to promote the recovery of the patient.

The remedies that have been mostly employed in cholera are bleeding, opium, astringents, and stimulants, either separately or conjointly. With respect to bleeding, it may be stated, that there are two periods at which its use has been advocated. These are,—(1.) At an early period, to diminish the quantity of black blood in very robust individuals, which would oppress the system at the more advanced stages of the disease. But even when performed early it will not prevent collapse: it is believed, however, to increase the chances of recovery from collapse. It is a highly dangerous remedy when attempted after the pulse has begun to sink, as it then induces an immediate and fatal collapse. (2.) Bloodletting has been advocated at a later period, to lessen the over-excitement of the central circulation, to moderate the violent action of the heart, and to diminish the quantity of black inarterializable blood coming from the more remote vessels. Most benefit is derived in this stage from opening a vein in the foot, or applying as many leeches to the foot as will be sufficient for the end in view (Dr. ANDREW BUCHANAN).

On the appearance of cholera in Europe, opium was administered indiscriminately in the doses recommended by the Indian practitioners, to the greater part even of an ounce of laudanum; but it was soon seen that, in the cold stage, it was inefficient in controlling the vomiting or purging, that it did not allay the spasms, and moreover, hardly produced any narcotic effect. The action of the accumulated doses of opium, though suspended during the cold stage, was often fully developed in the cases that lived to the period of reaction, and occasioned so much affection of the head, inducing fatal narcotism, or at the least, interfering with the functions of the kidneys, and so

leading directly to uræmic poisoning, that most practitioners either abandoned its use, or limited it to a mere fractional dose of that usually given in India—namely, from *three* to *twelve* minims of the tincture of opium, or half a grain to a grain of solid opium every four or six hours.

In considering the treatment of cholera there are three periods to be provided for,—(1.) The period of diarrhœa which so frequently precedes cholera; (2.) The algide period, or collapse; and, (3.) Period of reaction.

(1.) *The Period of Diarrhœa.*—To check or arrest the diarrhœa is the practical result aimed at by a variety of formula. Those in which opium is the main remedy have acquired the most amount of confidence.

The management of a case embraces the following points,—(1.) The horizontal position of the body [with perfect rest]; (2.) The administration of opium, with or without cordial stimulants; (3.) The induction of perspiration.

The necessity for the horizontal posture of the patient is, that it aids the efforts of the circulative powers, which tend to weakness.

With regard to opium, its dose must be regulated by (1.) The extent of the nervous prostration; (2.) The rapidity of the dejections; (3.) The extent of vascular depletion. In the cases which present these phenomena in the extreme a much larger dose of opium is required to be given at one time than in the cases less urgent at the outset.

The following formula for pills, each containing a grain of opium, with stimulants, is well known as an anti-spasmodic pill in the early stage of bowel relaxation:

R. Pulv. Opii, gr. xij; Camphor, gr. xxx; Pulv. Capsici, gr. ix; Spt. vin. rect., q. s; Conserv. Rosar, q. s.; Misce et divide in pil xij.

Moderate doses of opium or morphia, either alone or combined with stimulants, as the *pulvis cretæ aromaticus cum opio*, were often sufficient to check diarrhœa. The following (*cholera mixture*, as it was called) was proposed by the Board of Health during the prevalence of cholera, and was no doubt useful in many cases of diarrhœa:

R. Pulveris Aromat., ʒiij; Tinct. Catechu, fʒx; Tinct. Cardam. Comp., fʒvi; Tinct. Opii, ʒj; Mist. Cretæ preparat. ad fʒxx. Of this mixture the dose is one ounce.

Bulky doses of remedies are, however, very obviously objectionable; and the usual remedies known as “astringents” (compared with each other, or with opium) have no decided influence for good. “Astringents,” as such, have merely a negative effect.

[The prodromic diarrhœa is said to be promptly arrested by the *lavage* *Caillard*, composed of 19 parts of the sulphate of soda, and one part of common salt in a suitable quantity of water. *Sulphurous acid* has

proved efficient. Equal parts of paregoric and aromatic spirits of ammonia, to which a small quantity of camphor is added, is an excellent remedy.]

The extensive experience of my friend, Dr. Fergus, of Glasgow, in whose practice I was privileged to assist during the epidemic of 1849, led him to the conclusion, which I believe to be true, that "there is a first stage at which cholera is curable and preventible"—namely, laxity of the bowels previous to vomiting, spasms, or uneasiness of any kind. The relaxations of the bowels may not even amount to diarrhœa; and may be to the extent of only two or three stools a day, where one only was usual. Such relaxation was generally thought of no moment, being attended with no pain; on the contrary, the evacuations often gave a feeling of relief. This is the only stage at which opium is to be given, and that in a full dose. At this stage, in combination with a stimulant, it is often of the highest value. It is *only* to be given "*if the evacuations are still bilious, the pulse fair, and the skin warm.*" When vomiting, purging, and cramps set in, it is too late for opium—its administration then is absolutely poisonous.

When medical men have charge of large numbers of people, as in the army, navy, prisons, workhouses, asylums, hospitals, and the like, it is incumbent on them to make frequent inspections of those under their care, and to seek out any cases of such incipient diarrhœa. Responsible officers should be made to take notice of those who go more than once a day to the water-closets at times when a cholera epidemic influence prevails. "In military practice," as Dr. Maclean justly observes (MS. notes to the author), "frequent inspection of the men is of cardinal importance. Every man in a regiment should be seen at least three times a day by some medical officer, who should also visit the various guards. By walking down the ranks at roll-call, and picking out the men who show the earliest symptoms, cases are thus caught in the stage of premonitory diarrhœa and saved."

The following rules were drawn up by Dr. Fergus for the management of large numbers of men, in the factories and offices of Glasgow, over which he had charge when cholera was epidemic:

- "1. Do not be afraid of cholera, or make it the topic of conversation. Fear and all the depressing passions are injurious.
- "2. Do not take brandy; it is not a preventive; and it does harm by disordering the action of the stomach and bowels.
- "3. Do not make any change in your usual diet, if it is simple and of easy digestion; take it moderately, and at regular intervals, as long fasting is injurious; and carefully avoid excess in any intoxicating beverage.
- "4. Take no excessive fatigue; if overheated, beware of any sudden chill, and see that the skin is kept comfortably warm. If the disease appears in winter, much benefit may be derived from wearing a flannel belt around the body, covering the stomach and bowels.
- "5. As soon as cholera appears in a town, a bottle of solution of morphia or laudanum (and a graduated measure) should be kept in every house, place of business, factory, or wherever, in fact, there are a number of people gathered together. Persons travelling should always have it

with them, or easy of access. During the existence of the epidemic, one person in each factory, &c., should take the charge of the health of the inmates, and should act as 'house physician,' warning all under his or her care to attend to the slightest relaxation of the bowels. He should remind them that the less pain the more danger, and therefore the more need of immediate and energetic action. He should, if possible, ask every individual as to the state of his bowels two or three times a day.

"6. Should the slightest diarrhœa occur, the individual so attacked should at once receive forty minims of solution of morphia or laudanum.* If from home or at business, he should be at once conveyed home in a cab, put to bed, and kept warm. If chilled, warm water bottles may be put to the feet. If the first dose has not checked the looseness, the patient should take a second, and then have a flannel cloth thoroughly dipped in turpentine, placed all over the stomach and bowels for from forty minutes to an hour, or a large, soft, warm poultice of linseed meal and mustard for one or two hours. If the second dose has not effectually checked the diarrhœa, and medical assistance has not arrived, a third dose may be taken.

"7. The patient must remain in bed two or three days after the diarrhœa is checked. I *insist strongly on this*, for the patient often feels so well that it is difficult to get him to attend to it.

"8. To relieve the thirst, a piece of ice may be given, or a mouthful of iced water, or soda water, but in no case must more fluid be taken at a time, and all food should be abstained from till from fifteen to eighteen hours after the opiate has been administered. *Then*, and for two or three days, the diet should consist of such food as rice, sago, arrow-root, Indian corn flour, tea and toast, &c.; about the third day beef-tea or chicken-soup might be taken.

"9. These rules are for the *first stage*, and for it only—i. e., the diarrhœa. If a person has neglected the first warning, and is in the second stage—i. e., has cramps, vomiting, and stools like rice-water, without smell—you should, till medical assistance arrives, place the patient in bed surrounded with bottles of hot water, and give him a little ice, and mouthfuls of soda and water. If the cramps are severe, you must rub the limbs with turpentine, or chloroform and oil" (*Glasgow Med. Journal*, 1866).

But there are certain cases in which, although the diarrhœa may be altogether checked by such remedies, yet the disease is not cured. Symptoms characteristic of the *algide* stage and collapse supervene. These are the cases which give support to that method of treatment which has for its object elimination by the promotion of purging and of vomiting—excretion of the poison by the alimentary canal. In support also of this method of treatment, its advocates lay stress upon the fact that those are the worst cases in which the diarrhœa is the least; and that those cases are most hopeful in which diarrhœa and vomiting are the most severe.

Dr. Johnson's treatment by castor oil has for its object the elimination of a poison; and it may be said of it, at the outset, that it

* If a measure is not at hand, a small teaspoonful. Of course this dose is for adults. Below that age the doses should be a drop for each year, till twelve or fifteen, and after fifteen a drop and a half for each year, up to forty minims, or a small teaspoonful. More portable than laudanum, and of equal efficacy, would be pills composed of a grain and a half of opium and a grain of cayenne pepper in each pill, three of which may be taken with safety, till medical assistance arrives.

is neither more or less successful than other remedies of its class. It, as well as purely astringent mixtures, excite such loathing in most cases that they cannot be persisted in.

Dr. G. Johnson agrees with the rule that "diarrhœa during an epidemic season ought not to be neglected even for an hour." He regards such diarrhœa as an indication of the presence of offending material in the alimentary canal—*e. g.*, (1.) Unwholesome undigested food; or, (2.) A large and unnatural accumulation of the feculent contents of the bowel; or (3.) Noxious secretions poured from the blood into the bowels, in consequence of the action of a specific blood poison: to this latter Dr. Johnson believes *choleraic diarrhœa* to belong.

Therefore, he lays down the following rule,—“Not to attempt by opiates, or by other directly repressive means, to arrest a diarrhœa while there is reason to believe that the bowel contains a considerable amount of morbid and offensive material; for such must come forth before diarrhœa can permanently cease.” Purging he considers the natural way of getting rid of the irritant cause; and the safest purgative he believes to be castor oil. So far as meeting the conditions of examples (1) and (2) as above, the treatment cannot be improved upon; but that it is the best possible treatment to meet the preliminary diarrhœa or relaxation of the bowels in cholera cannot be regarded as established, nor warranted by what we know of the pathology of the disease as set forth in the text.

Opium is the remedy which, by actual experience, seems most worthy of reliance; *but only at the commencement of laxity, or relaxation of the bowels*, in seasons when cholera is epidemic.

On the treatment based upon conflicting theories as to the nature of the disease Dr. Fergus makes the following remarks:

“The influence of the theories of Dr. Johnson and others is to be seen in the instructions recently issued by the Board of Supervision in Scotland—very excellent as to the sanitary part, but in the medical portion (like most things issued by a committee) an evident compromise. It is a compromise, I believe, between the facts and a theory. . . . We are told, under the fourth section, to take castor oil or rhubarb and soda for looseness of the bowels, and afterwards the astringent mixture or pills—the former being intended as *eliminators* to promote the diarrhœa, the latter to check it. If this advice is followed during an epidemic of cholera, the consequences may be very serious. It is well known that during an epidemic there is a general tendency to relaxation of the bowels, and that they become very susceptible of the smallest dose of even the mildest medicine. Any one who has had much to do with cholera must have frequently met with cases which appeared to be the direct consequence or result of a slight dose of medicine. The risk is that the castor oil and rhubarb would drive most of the cases into the second stage of cholera, and then the use of astringents and opiates at that stage would increase the danger immensely.

“As to the remedies ordered, the quantity of laudanum (*viz.*, five drops per dose, is too small, and there is no necessity for the chalk and catechu. Before the opiate could produce much effect, the stomach would be overloaded with the chalk and catechu, to the extent of indu-

cing vomiting. The pills should be ordered after each discharge from the bowels; but people will find it easier to provide themselves with laudanum than to use complicated mixtures or pills."

[Dr. Leclerc, of Tours, France, and Dr. A. Rodrigues Barraut, of Mauritius, claim to have had large success with the following treatment. The extract of belladonna is given every half hour in quarter-grain doses, and continued until its physiological effects are produced, then increase the intervals to every 1st, 2d, 3d, and 4th hour, giving the remedy until the urinary secretion reappears. Atropia, used hypodermically, was found to relieve the cramps. White of eggs well diluted with water was freely given as a drink. Dr. Hodgen, of St. Louis, who has adopted the theory, and followed the treatment of Dr. Leclerc and Barraut, speaks well of the results. It has also been tried by Dr. J. W. Brewer, U. S. A., and he records a favorable experience.]

In the second or algide stage the object is to promote reaction and to keep it in moderation. If the patient is not seen till profuse discharges—rice-water-like—have taken place, the time for all active treatment has passed, and efforts must be directed solely to restoration and repair.

To promote reaction in cholera and diarrhœa, the following formula has met with most universal approval in this country and in India. So highly is it valued, indeed, that it is ordered to be always in store, and in readiness in the "*Medical Field Companion*" of the army when on the march:*

R. Ol. Anisi., Ol. Cajeput, Ol. Juniper, āā ʒss.; Æther., ʒss.; Liquor Acid. Halleri, ʒss.; † Tinct. Cinnam., ʒij; misce. *The dose of this mixture is ten drops every quarter of an hour in a tablespoonful of water. An opiate may be given with the first and second dose, but should not be continued, for reasons already given.*

Some physicians think calomel should be given in moderate doses, for the purpose of producing a flow of bile into the intestines, as well as of restoring the other suppressed secretions. The indications, however, more generally followed are to treat the case as we should a similar state in typhoid fever (and calomel in small doses has been shown to be of service in connection with the affection of Peyer's glands in that disease), and to moderate the affections of the bowels by mild opiates, by enemata, and by sinapisms to the abdomen; also, to relieve the head by leeches and cold lotions, and subsequently, as the tongue becomes brown, to support the patient with wine, sago, strong broths, and a generally cordial treatment.

[There is much evidence in favor of the administration of calomel in large doses (20 grains), frequently repeated, to control the vomiting and purging.]

* A memorandum from Savory and Moore, of date 7th June, 1866, shows that the quantities of the *essential oils* in the mixture now issued are increased to ʒiiss.

† The *Liquor* or *Elizir Halleri* consists of one part of concentrated *Sulphuric Acid* to three parts of *Rectified Spirit*. It is commonly employed in Germany in the treatment of typhus and allied diseases, in doses of five to twenty drops in solution (MURCHISON, *l. c.*, p. 266).

During the reaction stage Dr. Andrew Clark, of the London Hospital, employed, with great success, a powder composed as follows:

Hyd. c. Cretâ, gr. ij; Pulv. Ipecac., gr. ss.; Pulv. Doveri, gr. ijss.; night and morning.

The cases most benefited by this remedy were those in which the tongue had become dry and hard, saline, lemonade, and chlorate of potash drinks being freely given. Mustard plasters or blisters to the nape of the neck were believed to be of use by inducing the return of the urinary secretion.

[It is important to restore and maintain the urinary secretion, uræmic poisoning being one of the chief difficulties met with after the patient comes out of the stage of collapse. Ten grains of the acetate of potash and half a fluid drachm of the spirits of nitre may be frequently given in this condition.]

The sick-room should be supplied with fresh air. Liquids should be assiduously applied to every surface capable of absorbing them, and the patient should be suffered to remain as free from officious treatment as possible. Heat applied in the dry form is to be avoided, but cloths moistened with hot water may be applied; or the patient may be wrapped up in warm moist blankets, and hot bottles or bags of heated sand placed around his cold and benumbed body.

After the temperature is restored, the surface should still be kept moist, by sponging from time to time, or by the use of the wet sheet, to moderate reaction.

Urgent thirst is one of the most distressing symptoms in cholera. There is incessant craving for cold water—doubtless instinctive, to correct the inspissated condition of the blood, due to the rapid escape of the *liquor sanguinis*. It was formerly the practice to withhold water—a practice as cruel as it is mischievous. Water in abundance, pure and cold, should be given to the patient, and he should be encouraged to drink it, even should a large portion of it be rejected by the stomach; and when the purging has ceased, some may, with much advantage, be thrown into the bowel from time to time (MACLEAN).

Dr. Andrew Buchanan, of Glasgow, recommends the following as a drink generally relished and retained upon the stomach; "A raw egg beat up with half a pint of milk, and then mingling them with about a pint and a half of water, adding as much salt as will give the whole an agreeable taste." Or whey, milk and water, weak chicken soup, or any similar decoction, may be drunk *ad libitum*. *Enemata* of warm milk, repeated as often as expelled, are of the highest importance, by supplying an absorbable fluid, by mitigating abdominal pains, and diluting acid secretions.

The secretion of urine may be promoted by dry cupping over the loins, by the use of solutions of *chlorate of potash*, and the like. But suppression of this secretion is most to be dreaded where opium has

been too freely used in the treatment. In men of intemperate habits we often see, during the stage of reaction, obstinate vomiting of thick, tenacious, green paint-looking matter, probably bile-pigment, acted on by some acid in the stomach or alimentary canal. It is a symptom of evil omen, and often goes on uncontrolled until the patient dies exhausted, and this although all other symptoms may promise a favorable issue. It may last for a week, resisting all remedies, and proving fatal when the urinary secretion has been restored, and all cerebral symptoms have subsided. Alkalies in the effervescing form, free stimulation of the surface, and chloroform in small doses, offer the best hope of relief for such cases. The patient should be nourished more by the bowel than the stomach when vomiting is present.

Ice should be given *ad libitum*, where it can be obtained, not only to dissolve in the mouth, but to swallow in pieces of convenient size (MACLEAN).

A plan, peculiar perhaps to this country, and which was practised to bring about reaction when the inefficiency of medicines was generally admitted, was an injection into the veins of the suffering patient of a solution of half an ounce of *muriate of soda*, and of four scruples of *sesquicarbonate of soda*, in ten pints of water, of a temperature varying from 105° to 120° Fahr. This solution was injected slowly, half an hour being spent in the gradual introduction of the ten pints, and the immediate effects of this treatment were very striking. The good effects were rapid in proportion to the heat of the solution, but a higher temperature than what is stated could not be borne. After the introduction of a few ounces, the pulse, which had ceased to be felt at the wrist, became perceptible, and the heat of the body returned. By the time three or four pints had been injected, the pulse was good, the cramps had ceased, the body, that could not be heated, had become warm, and instead of a cold exudation on the surface, there was a general moisture; the voice, before hoarse and almost extinct, was now natural; the hollowness of the eye, the shrunken state of the features, the leaden hue of the face and body, had disappeared; the expression had become animated, the mind cheerful, the restlessness and uneasy feelings had vanished; the vertigo and noises of the ear, the sense of oppression at the *præcordia*, had given way to comfortable feelings; the thirst, however urgent before the operation, was assuaged, and the secretion of urine restored, though by no means constantly so. But these promising appearances were not lasting; the vomiting continued, the evacuations became even more profuse, showing that the remedy did not touch the root of the evil. Perhaps, if Hallier's observations are correct, it supplied a pabulum for the fostering and development of the mischief. The patient soon relapsed into his former state, from which he might again be roused by a repetition of the injection; but the amendment was transient, and the fatal period not long deferred. Of 156 patients thus treated at Drummond Street Hospital, Edinburgh, under the direction of Dr. Mackintosh, only twenty-five recovered,—a lamentably small

proportion, and, small as it is, it seems doubtful if the recoveries were final or complete (Mackintosh, *Principles of Pathology*, p. 365).

Stimulants tend to inflate the stomach, and are of no use in exciting the heart to any salutary action. Employed as in typhus, they are worse than useless.

All violent remedies in this disease, as in others, are greatly to be deprecated. Strychnia, prussic acid, tobacco, galvanism, boiling water, the actual cautery, or firing the spine, or bags of ice to the spine, or injections of spirits, are proven by trials to be of no use, nor can their recommendation be supported except upon baseless theories, having no rational relation to the pathology of the disease. Large doses of calomel or of lead are alike detrimental. Nature effects a cure by slowly restoring the constitution of the liquids of the body.

To generalize on the subject of remedies in cholera, the broadest conclusion seems to be that remedies with an acid rather than an alkaline or neutral reaction have been most beneficial.

Prevention of Cholera.—The following summary by the highest authority on this subject, Dr. Parkes, will best conclude this account of cholera. The importance of the topics noticed may justify, it is hoped, the amount of space which the subject has taken up:

"For the first time in the history of cholera a new system of prevention has been brought largely into play in Europe—viz., the addition to the discharges of a presumed disinfecting substance. This plan, of course, is based on the belief that the principal (perhaps the only) mode of spread is by means of the putrefying evacuations; and the results obtained by it give certainly some strong evidence in favor of this opinion.

"In this country the difficulty has been to make the public (and, in some cases, even the medical men) sensible of the importance of this plan, and of the necessity of giving it a complete trial. In some cases in which it has really been fairly tested, it appears to have arrested the spread of the disease, as at Bristol, and Dr. Budd's paper in the *British Medical Journal*, April 13, 1867, gives good evidence on this point. In Southampton also, I believe, the spread was limited in this way, though it was not arrested so perfectly as at Bristol. In London and several provincial towns the method was also tried more or less fully.

"In Germany, owing to the influence of Pettenkofer, the disinfecting plan was also brought into play, and the system followed has been described by that chemist.*

"Without analyzing all the evidence, I proceed to give the most important practical rules.

"The dangerous period of the choleraic stools is supposed to be when they become very ammoniacal. This occurs sometimes immediately they are passed, but usually not to any extent for some time. It is thought (but of course exact scientific proof is not readily attainable) that anything which makes and keeps them acid prevents the changes which cause the poison.

"The three principal means of doing this are the use of *carbolic acid*, and *sulphate of iron* (with or without *permanganate of potassium*), and

* "Cholera Regulativ, von H. Griesinger, M. Pettenkofer, and C. A. Wunderlich, *Zeitschrift für Biologie*, Band II, p. 435."

the *salts of zinc*. Each has its advantages, and all may be used. The *carbolic acid*, from its liquid form and from its volatility, is excellently adapted to purify air, and to be used when surfaces are to be washed. It is also useful for sewers and closets. The *sulphate of iron* in substance and strong solution is better adapted for being put in the utensils in a room, as it has no smell, but it may be equally used for sewers and for watering streets. The *sulphate of zinc* (for the *chloride* is too dear) is better adapted for being put on linen or on floors, as it does not iron-mould the linen like the *sulphate of iron*.

"The *carbolic acid* has not been used much in Germany, as it is still too dear; but Pettenkofer makes an observation of importance—viz., that when added to *sulphate of iron* the mixture seems to have more power of preventing ammoniacal development than either substance separately. If so, it might be desirable, as a matter of practice, to use the two together as much as possible. The salts of zinc (*sulphate* or *chloride*) may be also used, but are perhaps not so good, and in some forms are dearer than the iron salts. *Chloride of lime* does not prevent the ammoniacal change, and appears altogether less useful.

"The quantity in which these substances must be used is as follows: For each healthy person, daily, about three-quarters of an ounce of *sulphate of iron*, or one drachm of strong (but impure) *carbolic acid*, are sufficient. This amount will entirely prevent any decomposition of the *feces* for several days. In a town, therefore, where sewers are used, the above amount of *sulphate of iron* or *carbolic acid*, multiplied by the number of persons, should go into the sewers daily, and, if possible, should be passed in from the houses, so as to act on the house drains as well as on the main sewers. If the place is not sewered, then the disinfectants should be added to the cess-pools, middens, latrines, or whatever plans may be in use. If both *sulphate of iron* and *carbolic acid* are used, which is to be recommended, half the quantity of each should be employed. The iron should be dissolved in a good deal of water.

"Dr. Kühne, who has made a great number of experiments on the action of various agents on fermenting substances, does not reckon the value of the *sulphate of iron* or of *carbolic acid* so highly as other observers. He states that neither arrest the various fermentations. Such an arrest, is, however, attained with strong alkalies and strong acids; with *chlorine*, *chloride of lime*, *bromine*, *permanganate of potassium* and *sodium*, and *permanganic acid*. On the hypothesis, therefore (for it is nothing more), that the dangerous condition of the cholera discharges is one of 'fermentation,' he recommends any one of these substances rather than *carbolic acid*, and for common use prefers *permanganate of sodium*, to which (as a concession to Pettenkofer) he adds some *sulphate of iron*. The proportions are two parts of *permanganate of sodium* (solution?), forty-five parts of *acid sulphate of iron*, and fifty-three parts of water in one hundred parts.

"It must be remembered, however, that such points as these must be decided by actual experience, and that arguments derived from the action of these substances on common ferments are not very satisfactory as regards the prevention of cholera.

"In Southampton, in 1866, carbolic acid was chiefly used; and the average amount was about twenty gallons daily for a town of 50,000 people: it certainly appeared useful.

"If an aerial disinfectant is needed, *sulphurous acid* (obtained by burning sulphur) is perhaps the best. *Nitrous acid fumes* are certainly

very powerful; and one or other of these substances should be used for half an hour daily in all privies or latrines.

"For washing clothes the iron salts are not applicable, as they stain linen. *Carbolic acid* gives a disagreeable smell. Either a watery solution of *sulphurous acid* or a solution of the zinc salts should therefore be used. Baking the clothes, at a temperature of 250° , or boiling, should be used.

"In hospital wards, dead-houses, &c., it is a good plan to sprinkle sawdust on the floors, and to moisten it with weak *carbolic acid* (one part of crude acid in sixty or eighty of water).

"These measures should be commenced when cholera is apprehended. Every privy and sewer should have twice daily the mixed *carbolic acid* and *sulphate of iron* solution. If cholera is introduced, the amount should be doubled in the privies of all the adjacent houses, while the closet of the affected house should never act without a portion of the disinfecting liquid being placed in it. If the disease breaks out, a plan recommended by Dr. Budd is worthy of imitation—viz., to place a layer of *carbolic acid* powder (carbolic acid and lime) in the bed, under the breech of the patient.

"The disinfection in this way of the closets and privies of hotels, railways, and workshops, should be commenced very early.

"As a precaution against cholera, quarantines have only answered when they are absolute, and an absolute quarantine is not possible for a commercial people. The reason of the failure of partial quarantines is the fact that the diarrhœal stools will propagate the disease, and that the period of incubation, though usually short, may be prolonged even to twenty or twenty-five days. Restriction on movement must therefore be used or not, according to circumstances, but in all cases persons coming from infected districts ought to take measures for disinfecting their evacuations in the above manner."

The inquiries made through the Epidemiological Society show that special hospitals, or special wards in hospitals, ought to be devoted (with a separate and special staff of attendants for day and night duty, or more frequent periods of relief) entirely to the management of cholera patients.

For further details, see management of epidemics generally, page 226 *ante*.

[The possibility of cholera reappearing in the following year at places visited by it during an epidemic, if the most stringent hygienic precautions are not adopted, should be borne in mind. The effect of the rigid enforcement of proper sanitary measures in hindering the spread of cholera is conclusively shown in the Army Reports, to which reference has been made, and to which the reader is referred.]

[CHOLERA MORBUS—*Sporadic Cholera, Cholera Biliosa.*
(DR. CLYMER.)

Definition.—*A disorder of hot climates and hot seasons, characterized by sudden, incessant, violent vomiting and purging of acrid matters, sometimes bilious; attended with spasms of the muscles of the abdomen and extremities, cold surface, feeble and rapid pulse, and prostration which may pass into collapse; the direct exciting cause being generally some undigested article of diet.*

History.—The term *cholera* has been in use since Hippocrates (*Epidem.*, lib. v). Celsus derives it from *χολή*, *bile*, and *ῥέω*, *I flow*,—a *bile-flux*; others from *χολός*, *intestine*, and *ῥέω*—*intestinal-flux*; whilst Trallian and Ruysch give as its derivation *χολήρα*, *the rain-gutter of a house*. Galen gives its true pathogeny. Celsus accurately describes it (lib. iv, cap. 2), and mentions, as an occasional symptom, watery and white discharges; he speaks of it as a dangerous disorder, and one that may quickly cause death. Aretæus (lib. ii, cap. 5) is minute in his account of the gastro-intestinal evacuations. Of modern writers Sydenham has given a most graphic delineation in his narration of the disorder as it prevailed in England in 1669.

Nature and Pathogeny.—Cholera morbus is a disorder of hot climates, and the hot seasons of temperate climates. Sydenham says, "It seems partial to a particular part of the year. It sets in at the end of the summer and beginning of autumn, as truly as the swallow comes in spring, or the cuckoo sings in summer."* It is most common in that part of the hot season in temperate countries when the temperature during the day is high and falls at night-time; or after those sudden weather-changes so frequent in our summers. Whatever part climate and temperature may have in its causation, it is only collateral and predisposing, the immediate cause in most cases met with in practice being the presence in the stomach and upper bowel of some article of diet, imperfectly digested, which sets up a fermentative and putrefactive process, making the matters acid and acrid, and causing great irritation of the gastro-duodenal lining membrane, which brings on vomiting and purging.† The irritation extends to the liver, inciting, in some cases, the biliary secretion, for a while,—to the spinal cord, causing cramps in the muscles of the abdomen and legs,—to the sympathetic, producing, through the periphoro-vascular system, coldness of the skin and capillary torpor. The most common offending articles of diet are shell-fish, salted or tainted meat, and decaying vegetables. From the peculiar state of the digestive organs, sound food may not be acted on by the gastric juices, and, undergoing the putrefactive process in the stomach, becomes an exciting cause. Drinking largely of iced-water, when the body is overheated, is an occasional cause. Emanations from cesspools and sewers, and putrilage, have produced a train of symptoms identical with those of cholera morbus.

Cholera morbus is a disorder essentially distinct in its nature and ætiology from Asiatic cholera. The one is caused by a material toxic agent directly in contact with the gastro-intestinal membrane, rendered specially susceptible to its action from the influence of season and temperature; the other owns for its cause a specific poison, infecting through the atmosphere and the evacuations of those suffering from it. The discharges of cholera morbus are acrid, dark-colored, and, often, during some time in an attack, bilious; while in Asiatic cholera, there is no evidence of the presence of bile in the evacuations, and they are light-colored, and turbid from whitish particles—the epithelial cells of the mucous membrane of the stomach and bowels.

Symptoms.—An attack of cholera morbus is most often sudden,—though it may be preceded by nausea, colicky pains, and rumbling in the belly—beginning with incessant, uncontrollable simultaneous vomiting and purging; first of the usual contents of the stomach and bowels, and

* Med. Obser., ch. ii. Works of T. Sydenham. Syd. Soc. ed., vol. i, p. 163.

† Galen attributes cholera morbus to the presence of acrid humors generated by the corruption of the food.

then of highly acrid, acid, and often bitter matters, varying in color from a light brown to a dark green. The discharges both from the mouth and anus are suddenly and violently projected, rocket-like, or squirted out without effort. The stools are very fetid. At the outset there may be tympany, which soon disappears, the belly becoming contracted. Painful and quick recurrent cramps in the abdomen, legs, and, sometimes, upper extremities, come on, the muscles contracting into hard knots. The thirst is excessive, and there is a keen craving for drink; the tongue is covered with a whitish or yellowish fur. The pulse, depressed at first, becomes quick, compressible, and gradually feebler; the skin very soon gets cold, and is bathed with moisture; the voice is weak, and its tone peculiar; the breathing is oppressed and sighing; the urine is high-colored and scanty; and debility is complained of. If the disorder is not checked these symptoms worsen, the features become shrunken, the eyes have a hollow look and are surrounded by bluish circles; the skin is shrivelled, algid, blue, and covered with a clammy sweat; the pulse is fluttering, and finally imperceptible; the respiration labored; the secretion of urine stops; the discharges and spasms cease, though there may be retching and a desire to go to stool; hiccough is troublesome; the prostration is great and growing; collapse is perfect; and finally death happens from asthenia. The mind is usually throughout unaffected both in mild and in severe attacks. A favorable issue, the ordinary one in this country, is shown by diminished violence of, and longer intervals between, the spells of vomiting and purging, a more natural look, and gradual reaction.

The *duration* of an attack varies from a few hours to twenty-four; it rarely lasts several days. Recovery is usually rapid, though symptoms of gastro-intestinal irritation may remain for some days, and there is not unfrequently much emaciation and haggardness even after a short sharp attack.

Diagnosis.—The diacritic features of cholera morbus and epidemic cholera have already been dwelt upon. There is much more likeness between the symptoms of cholera morbus and those produced by irritant poisons, but a differential diagnosis is generally easy; the history of the case, when it can be got, and an examination of the vomit and the state of the mouth and fauces, where the poison is a caustic, should prevent a mistake.

CHOLERA MORBUS.

After vomiting and purging, sometimes, scald of the mouth and fauces complained of.

Vomiting and purging often simultaneous.

None, or slight pain in epigastrium.

Cramps in abdomen and legs.

Never.

POISONING FROM IRRITANT SUBSTANCES.

Burning heat in mouth, pharynx, and œsophagus usually precedes vomiting, and is more intense.

Vomiting generally precedes purging, and is not simultaneous with it; and matters are ejected with less force.

Constant severe pain in epigastrium.

None.

Alvine evacuations often bloody.

Treatment.—Opium and its preparations must be our chief trust in the treatment. They are to be given both by the mouth and rectum, in liquid form, and in full doses, to be repeated at once if thrown off, and if kept, at such intervals as to hinder narcotism. There is great tolerance of opium in this disorder. Morphia may be used hypodermically.*

* [From a limited experience with the sulphites in cholera morbus, the writer is disposed to think that, in small doses, at the outset of an attack, they may prove beneficial.]

A chief point is to withhold all drink, but small pieces of ice may be held in the mouth, the water being spit out. Dry rubbing with the hand, or with woollen cloths, should be made over the body, and particularly over the abdomen and lower extremities, to relieve the cramps, and revive the capillary circulation. Sinapisms may be applied to the calves of the legs, inside of the thighs, and along the spine, in severer cases; or the upper and lower extremities, belly, chest, and spine, be covered with woollen cloths, well wrung out of hot water, to which mustard flour has been added, and the patient then wrapped in a dry warm blanket. If prostration is very great with a tendency to collapse, a mixture of chloroform, camphor, ether, and capsicum, will prove an excellent stimulant, and has sometimes quickly checked all the symptoms. Iced brandy and iced champagne are favorite stimulants in an advanced stage of the disorder. On the abatement of the acute symptoms, the tongue remaining furred, a mild mercurial, followed by a small dose of castor oil, may be administered without risk of bringing on a relapse.]

[CHOLERA INFANTUM—*Summer Complaint—Infantile Cholera.*

(DR. CLYMER.)

Definition.—*A perilous disorder of early infancy, most common in the first year of life, the chief symptoms being stubborn purging of variously colored serous fluid, and vomiting; occurring under the combined influence of high atmospheric temperature, great humidity, and malnutrition. The solitary follicles of the intestines are more or less diseased.*

History.—Cholera infantum has been thought by many writers to be a disorder peculiar to the United States, but, as Dr. Trousseau remarks, it has always been observed, and shows itself in all countries under the same circumstances of season, age, and bodily state, and he describes it as *cholera infantile* or *mal d'été*.^{*} Sydenham says, "There is a sort of cholera morbus exceedingly fatal to infants."[†] It is probably more generally prevalent in the Middle and Western States than in Europe, recurring with uniform seasonal regularity, and being the chief cause of the great loss of life amongst infants in the large towns of those States during the summer months. There is no doubt that in the death-records distinct pathogenetic affections are ignorantly reported under the same general head of cholera infantum,—as the diarrhœa of dentition, cholera morbus, enterorrhœa, enteritis, and typhoid fever; yet we must admit the existence of a disorder of early infancy, of a special nature, characterized by typical symptoms, bred by the operation of the same causal factors, and whose true pathogeny and precise phenomena have yet to be accurately studied and described.

Symptoms.—One form of cholera infantum resembles very much the cholera morbus of adults; is sudden in its invasion; and has for its chief symptoms incessant and violent purging and vomiting of serous fluid, of a greenish or yellowish hue; great thirst; sharp and incessant cries, betokening pain; cold surface; quick pulse; and early collapse. Such cases may end fatally within twenty-fours. The more chronic and common form of the disorder begins with looseness of the bowels, soon followed by steady vomiting. The intestinal evacuations are at first fecal, very liquid, sour and offensive, but soon become serous, of a light-yellow color—like water to which a little yolk of egg has been added, or

^{*} Clinique Médicale, 2d ed., t. 3, p. 128. Paris, 1865.

[†] Works of Thomas Sydenham, M.D.: *Processus Integri*, ch. xxiv, vol. ii, p. 267; *Syd. Soc. ed.*, London, 1850.

the urine—which changes often to a greenish hue, from the presence of shreds or flocculi, like finely-chopped spinach or sorrel. Occasionally the stools may be for a while pasty and frothy, with a more or less yellow or green tinge. Vomiting is rarely an initial symptom, but soon sets in, and is incessant; everything swallowed is thrown up at once: if milk has been taken it is rejected in cheesy lumps; the odor of the ejected matter is sour, and it is often quite acrid. In the early stage the temperature of the body is natural, or very slightly raised at times during the day; the rate of the pulse is quickened; the tongue is moist, and its base is coated with a light-brown fur; the belly is slightly tympanitic; the child is restless, irritable, and cryful. As the phases of the disorder deepen, there is rapid and, frequently, excessive emaciation; the surface is dry and of a dusky hue, with heat-exacerbations; the skin of the body and extremities is flabby and inelastic, a fold of it pinched up between the fingers remaining for some time; the pulse is wiry, sharp, small, and frequent; purging and vomiting continue. There is little change in the character of the stools, except the predominance of the greenish hue, owing to the presence of broken-up blood-discs, and dark granular coloring matter; sometimes they are pinkish, like water in which fresh meat has been washed; there is rarely any trace of bile in them after the first day or two; they are acid, and have a cadaveric smell. Vomiting, or the effort to vomit, is obstinate and distressing, and, on placing the hand over the abdomen, a spasmodic or rolling motion of the muscles will be felt, simultaneously with the action of the stomach, though sometimes preceding it. There is pain in the abdomen, which is retracted and hot, shown by a pinched and distressed expression of the face, by a fold in the commissure of the lips outside of the orbicularis muscle, and by a line extending from the labial angle to the inside of the ala nasi, and sometimes surrounding the orbicularis (STEWART). Thirst is ardent, and when any liquid is offered it is swallowed with avidity, the vessel being seized, or if the child is lying down, it will quickly raise its head, no matter how weak or indifferent it may be. The eyes are hollow, with a blue circle; the face is wan; on awaking there is knitting of the eyebrows followed by a loud and frequent cry; and the tone of the voice is changed. There is much debility from the outset, and it gradually increases and becomes excessive. At this stage of the disorder there may be collapse and speedy death, or cerebral epiphenomena may happen, as convulsions or coma. Not infrequently there is more or less stupor, the eyes drawn up beneath the upper lid, with injected conjunctiva, rolling of the head, hydrocephalic shriek, *tache cérébrale*, which end in deep coma, or convulsions, particularly if there is dental irritation. Or, sooner or later, and this is probably the most common termination, typhous symptoms, the necessary result of prolonged innutrition, set in, and soon carry off the little patient.

Causes and Nature.—It would seem necessary that certain conditions should come together for the production of cholera infantum; it is the sure sequence of given antecedents. The effective causes may be said to be *age, high temperature, humidity, and malnutrition*. The influence of age is positive, it being a disorder limited to the first eighteen months of life. Stewart asserted that the true disease never appeared but during the period of teething.* Of 278 reported deaths from cholera infantum during the week ending July 21, 1866, 199 were under one year. It is a disorder of the hot months. In the city of New York, during a period of

* Prize Essay on Cholera Infantum. By James Stewart, M.D. The Transactions of the New York Academy of Medicine, vol. i, part vi, 1856.

eleven years, out of 1245 deaths from cholera infantum, 1061 were in the months of July, August, and September. During the first twenty-eight days of July, 1866—a month of unusually great heat—there were reported 687 deaths from cholera infantum, in a total mortality from all diseases of 3452. In the preceding trimester, in a total mortality of 5597, the deaths from cholera infantum were 76. The influence of *high temperature* is more striking, if the weekly mortality of the month of July, 1866, from cholera infantum, is examined. For the week ending July 7, mean temperature $81\frac{3}{4}^{\circ}$ Fahr.: total deaths, 493; deaths from cholera infantum, 61. For the week ending July 14, mean temperature $82\frac{1}{4}^{\circ}$ Fahr.: total deaths, 827; deaths from cholera infantum, 172. For the week ending July 21, mean temperature, $82\frac{1}{2}^{\circ}$ Fahr.: total deaths, 1362; deaths from cholera infantum, 278. For the week ending July 28, mean temperature, $73\frac{1}{2}^{\circ}$ Fahr.: total deaths, 770; deaths from cholera infantum, 176.

The late Dr. James Stewart instituted a series of observations to ascertain the relation of *atmospheric humidity* to cholera infantum, and came to the conclusion that ordinary climatic humidity had but little influence in its development; but, directing his attention to the occasional state of the dew-point, as it occurred in localities where the disorder was most rife—the population living in overcrowded underground cellars and tenement-houses—he “discovered a great difference within doors between it and the general dew-point of the external air, continuing often for a long time.” His observations showed—(1.) That the moisture was always greater nearer the surface of the earth, the difference at times being 4° ; (2.) That in very hot weather, in crowded rooms at night, “when all were within, the dew-point is very nearly the temperature of the air, thus saturating it with moisture.” With a temperature of 90° to 95° , and a dew-point in a crowded room almost equal to the temperature, a feeling of suffocation is experienced, which is easily accounted for when it is known that the dew-point of the breath, as it is expelled from the lungs, is 94° , and that the mean dew-point of the atmosphere is 38° ; and also, that in the hottest weather it rarely exceeds 70° .*

Malnutrition is due to several sources. The depressing and exhausting effects of great heat and moisture are exerted on a body imperfectly nourished. The mother, suffering from the devitalizing and septic influences which surround her, secretes a milk imperfectly elaborated and unfitted for food; or, when in itself wholesome, the digestive organs of the infant are so disordered, that there is a lack of the changes it should undergo; a chemical act of decomposition happens, directly the opposite of the vital act of digestion, and cheesy clots of the coagulated casein are rejected in the vomit, or pass unaltered or corrupt through the bowels. Infants brought up by hand, as well as those just weaned, are very liable to cholera infantum. Here a deleterious and inadequate diet—often exclusively farinaceous or gelatinous, or of milk from diseased animals, or so dilute as to be unfit for food—plays a chief part. The direct influence of *animal poison* must not be passed over. The chief haunts of cholera infantum are the fever-nests and cholera-fields of large towns. Diphtheria, scarlet fever, putrid sore throat, typhus, and Asiatic cholera, herd together, and fester in the slums, where the wretched and squalid dwellers are stowed away in ill-ventilated, ill-drained underground cellars and tenement-houses, in the midst of every possible insanitary condition; breathing a septic atmosphere, poisoned without, by the putrilage of slaughter-houses, soap and glue factories, cesspools and sewers, and

* Loc. cit., p. 290.

within by emanations from the human body, filthy clothing and bedding, and often the excrements of man and beast.

The *necroscopic characters* are—congestive patches of the mucous membrane of the small intestines, sometimes extending to the large, which is covered with mucous or membranous exudations; the solitary follicles are invariably enlarged, often resembling the sprinkling of white sand over its surface, and frequently ulcerated (J. JACKSON, HORNER, BAXTER, LINDSLEY, HALLOWELL, STEWART). Many observers state the liver to be congested, enlarged, or altered in color or texture, it being lighter than when healthy, or variegated and firmer. In 37 cases, carefully examined after death by Dr. J. Lewis Smith, there was no evidence that the liver was affected, either as to size, or in any way involving any modification of function. Microscopic examination showed a variable amount of fat—sometimes in excess, sometimes deficient, and sometimes greater in one part of the organ than another. The green stools, so long referred to the liver, are due to causes operating within the intestines, the green color not appearing until the lower part of the jejunum, or upper part of the ileum is reached. The green matter under the microscope is found to be in small shreds or masses (*American Medical Times*, vol. v, 1862). Where coma and convulsions have happened, there are softening and hyperæmia of the cerebral substance (HALLOWELL).

The conditions under which cholera infantum is produced, the constant intestinal lesion, and the symptoms of the more chronic and common form of the disorder, bring it in close relation to chronic camp diarrhœa, and would place it, in its developed stage at least, amongst autophagic diseases. All the deleterious causes which act as factors in the one case are equally potent in the other. The greater the insanitary conditions of any given locality, the higher the death-rate from cholera infantum. The digestive organs, disordered by the circumfusa, are unable to perform their office; for days and weeks, at a time of life when the tissue-changes are active, the constant waste of the body is unrenewed; there is, besides, another and great destructive force at work in the large and frequent drain by the intestinal discharges; there are extraordinary demands and no supply. As a consequence, all the phenomena of innutrition occur,—rapid and excessive emaciation, wan and shrunken face, harsh, dry, and dusky skin, and general typhous symptoms, including the cerebral epiphenomena, when these are not directly due to the irritation of dentition.

Treatment.—The indications for the treatment of cholera infantum are clear and positive. The conditions which seem to be the likely cause of the disorder must be got rid of. The instant threatening symptoms are the purging and vomiting, showing the state of the stomach and bowels. These must be stopped. That there is an excess of acid in the stomach and bowels, at least in the early stages of the disorder, cannot be doubted. Fermentation, and not chymification, is going on in the stomach and duodenum, and is, without question, one of the causes of the vomiting and purging. Various remedies have been suggested and used to check these symptoms, as calomel in fractional doses, mercury with chalk, creasote, the alkalies, &c. Small doses of the bisulphites of soda or potassa, with limed whey, will often act very happily, whilst the effect of poisonous drugs is always doubtful, and generally positively harmful. Mercury cannot have any curative power; it is at best negative. If not contra-indicated by cerebral epiphenomena, and the effects carefully watched, opium and its preparations will be found a valuable remedy. Sydenham speaks strongly in its favor, and the prejudice against its use, is not, within certain restrictions, warranted. Flannel, wrung out of hot water, and on

which laudanum is poured, applied to the spine, will be found useful in checking vomiting. The skin, in common with all the excreting organs, is inactive. The effect of great and prolonged heat is always to increase its function; there is for awhile local hyperæmia, with swelling of the papillæ, giving rise to the eruption familiarly known as prickly-heat. Finally there is loss of tone and sluggishness, especially when exposed to currents of air; and its function must be excited. This may be done by gentle friction by woollen cloths, or a warm alkaline bath, in which the little patient should not remain longer than three minutes, and then be quickly dried, and wrapped in flannel. So soon as the stomach and bowels will tolerate food, it should be given of fitting quality, and in proper quantity. Farinaceous food (the carbo-hydrates),—the usual diet in all diarrhœal disorders,—is entirely inappropriate. The starches are unaltered by the peptic solvent, and turn acid in a mass; very often in the mouth, from the action of the saliva, so abundant in infants, before they reach the stomach. In infants so fed they will be found in the bowels almost unchanged (N. GUILLOT). The natural food of the infant is entirely dissimilar from these amylaceous articles, which not being digested and assimilated, are consequently not only innutritive, but act as direct irritants to the alimentary mucous membrane. Limed milk, to which a little gelatine has been added, or rennet whey, may be given; but in protracted cases, where the prostration is great, and emaciation rapid, beef essence, freshly prepared and well salted, will often be well borne and quickly appropriated. The avidity with which the little patient takes animal food*—seizing on meat, salt fish, &c., when it can,—shows an instinctive craving for proper nourishment, too constantly withheld from false notions of the nature of the disorder. Some twenty-five years since, Dr. Weisse, a Russian physician, used raw meat in a case of infantile chronic diarrhœa, and with success. It is a popular remedy in some parts of Europe in chronic and wasting affections. His example was followed by others, especially by Dr. Trousseau, of Paris.† Lean beef or mutton is first finely hashed, pounded in a mortar to a pulp, and then passed through a fine sieve; a thick concentrated juice—*purée de viande*—is thus obtained, nutritious and digestible, and often, when salted or otherwise flavored, quite acceptable. Give a half to three-quarters of an ounce, in fractional doses, the first day; and if well borne by the stomach, increase the quantity day by day, until a quarter or half a pound is taken in the course of the twenty-four hours. Dr. Trousseau mentions a case in his own family, where he continued it for more than one year; the child, about two years of age, finally taking a pound of raw meat daily. For the first day or two much of it may pass hardly changed in the stools, but this alone should not prevent it being persevered in. From the observations of Weisse, Braun, Van Siebold, and Trousseau, this diet, if long continued, is liable to generate tænia. White of eggs, thinned with natural or artificial Selters, or Vichy, or weak lime, water, is an excellent drink; or a few grains of bicarbonate of soda, may be added to the albuminate. Tonics and stimulants are very frequently required in the course of the disorder. Of the former, minute doses of arsenic (the liquor arsenici chloridi), alone or combined with quinine, or

* ["I have seen many children recover," said the late Dr. Rush, in reference to the treatment of cholera infantum, "from being gratified in an inclination to eat salted fish and meat. In some instances they evince an appetite for butter, and the richest gravies of roasted meat, and eat them with obvious relief to all their symptoms."—*Medical Inquiries and Observations*, 2d ed.]

† Clinique Médicale, par A. Trousseau, t. iii*, 2^e ed., Paris, 1865.]

the chloride of iron, or the perntrate of iron, or the tincture of nux vomica, may be given. Wine-whey, or brandy and water, to which a few drops of the aromatic spirits of ammonia have been added, are the best stimulants. The gums should be often looked to, and freely lanced if hot and swollen. But all treatment is too often unavailing, unless the child is removed from the insanitary conditions by which it is surrounded. The effect of change of climate is generally immediate and lasting, and where it can be done, the little patient should be sent without delay to a cool and mountainous region.]

CHAPTER VI.

PATHOLOGY OF THE ENTHETIC ORDER OF ZYMOTIC DISEASES.

THE diseases which belong to this order have the common property of becoming developed in the system after the introduction by inoculation or implantation of specific poisons. The sources of such poisons are more distinctly traceable than those which produce the miasmatic diseases; in other words, the substance or material which contains the *poisonous principle* can be obtained in most instances, although the *principle* itself has not been isolated by any chemical process. The poisons which produce the diseases of this order may be introduced through thin or abraded cutaneous surfaces, or through mucous membranes by the process of absorption, although, in most instances, it is believed that some solution of continuity exists. Others are directly introduced by weapons which inflict a wound or abrasion, and which at the same time introduce the poison. "Poisoned wounds" thus indicate or name a disease defined as follows: "*Wounds inoculated with foreign matter, producing general symptoms, or propagating inflammation to other parts of the body.*" In all instances the poison is received into the system by the processes of absorption, and the individual thus becomes inoculated. Thus, germs of a *specific* kind become directly implanted, and by a zymotic-like process become developed and increased in quantity or virulence till symptoms and effects are produced characteristic of the specific affections; and hence the name given to this order of diseases. No one has better illustrated the pathology of these diseases than Mr. Paget, from whose lectures *On Surgical Pathology* the following statement is given:

When a morbid poison is inoculated, it produces a specific effect, both on the tissue at the place of insertion and on the blood, as soon as the poison, or any part of it, is absorbed; in other words, it produces both a constitutional and a local change; and in both these effects its history must be traced. The specific local change is best seen in the implanting of certain animal poisons, such as those of venomous serpents and insects, and the results most rapidly follow the implantation of such poisons. The consequences of the insertion of such poisons are peculiar, and constant in their peculiarities. The bite of a bug, for example, is followed, within less

than a minute, by itching in the bitten part, and very soon a wheal, or circumscribed pale swelling, with a nearly level surface and a circumscribed border, gradually rises and extends in the skin. The swelling is produced by œdema of a small portion of the cutis at and around the bite. As the itching subsides, the pale swelling becomes less defined, and the more general vascular swelling of the surrounding and adjacent tissues gradually encroaches on the primary swelling at the bitten spot. In about twenty-four hours a papule or some form of secondary inflammation appears, with renewed itching at the site of the puncture. This, too, in the case of the bug-bite, gradually subsides. The primary swelling here described illustrates the immediate effects of the morbid poison on the tissue at and round the seat of inoculation, and within the area of such a swelling the tissues are, by the direct contact or influence of the venom, altered in their nutritive relation to the blood. Such specific alterations of the tissues at the seat of inoculation occurs with the syphilitic, the vaccine, and such-like virus; but the direct influence is most rapidly shown in the effects of the bites of the viper, the rattlesnake, and the *cobra di capello*. In such cases sloughing of the areolar tissue is established immediately after the bite. The poison seems to operate at once on the tissue, neither in the direction of the nerves, nor of the absorbents, nor of the bloodvessels; but the slough forms at the puncture, as if the venom had completely and at once killed the tissue (BRODIE, PAGET).

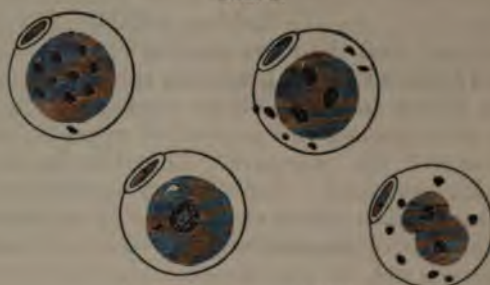
A secondary inflammation soon appears at the bitten or punctured part; and the occurrence of this new inflammation may be ascribed, in some measure, to an influence exercised by the virus on the blood; and it proves that the part does not return to health, although the first effects of the inoculation may subside. It proves that some material of the virus remains, or that the effects it has already produced upon the tissues at the injured part alter their relations to the blood, and render the part prone to specific disease. These specific effects upon the part may remain locally quiescent for a considerable length of time—during all that period of latency or incubation which intervenes between the inoculation and the appearance of the specific disease. But during all this interval—during all this period of incubation—the tissues at the site of inoculation are constantly changing; and the virus itself, like all organic matter, is probably in constant process of transformation till the *zymosis* is complete, and the specific disease is fully developed and expressed by various constitutional phenomena.

Dr. George B. Halford, Professor of Anatomy in the University of Melbourne, has recently (*Brit. Med. Journal*, July 20, 1867) given an interesting account of the action of the poison of the *cobra* upon the blood.

“When a person is mortally bitten by the *cobra di capello*, molecules of living ‘germinal’ matter are thrown into the blood, and speedily grow into cells, and as rapidly multiply, so that in a few hours millions upon millions are produced at the expense . . . of the oxygen absorbed into the blood during respiration; hence the gradual decrease and ultimate ex-

tion of combustion and chemical change in every part of the body, followed by coldness, sleepiness, insensibility, slow breathing, and death.

FIG. 2.



Magnifying power $\times 1050$.*

FIG. 3.



Magnifying power $\times 400$.*

The cells which thus render in so short a time the blood unfit to support life, are circular, with a diameter, on the average, of $\frac{1}{700}$ th of an inch. They contain a nearly round nucleus of $\frac{1}{2800}$ th of an inch in breadth, which, when further magnified, is seen to contain other still more minute spherules of living 'germinal' matter. In addition to this, the application of magenta reveals a minute colored spot at some part of the circumference of the cell. This, besides its size, distinguishes it from the white pus or lymph-corpuscle.

"Thus, then, it would seem that, as the vegetable cell requires for its growth inorganic food and the liberation of oxygen, so the animal cell requires for its growth organic food and the absorption of oxygen. Its food is present in the blood, and it meets the oxygen in the lungs; thus the whole blood becomes disorganized, and nothing is found after death but dark fluid blood, the fluidity indicating its loss of fibrine, the dark color its want of oxygen, which it readily absorbs on exposure after death.

"Let it not be thought that microscopic particles are unable to produce such great and rapid changes. It is well known, and I have frequently timed it with my class, that a teaspoonful of human saliva will, when shaken with a like quantity of decoction of starch, convert the whole of the latter into sugar in a little less than one minute. If ptyaline, the active principle of saliva, exerts this power at most in a few minutes, then surely the active principle of the secretion of the serpent's poison-gland may exert an infinitely greater power in as many hours.

"It results, then, that a person dies slowly asphyxiated by deprivation of oxygen, in whatever other way the poison may also act, and so far as

* Figs. 2, 3.—Changes in the blood-corpuscles subsequent to the bite of the *cobra di capello*.

the ordinary examination of the blood goes, the post-mortem appearances are similar to those seen after drowning and suffocation."

The changes which the absorbed virus undergoes in the living and infected body are,—(1.) Increase; (2.) Transformation; (3.) Combination; and (4.) Separation or excretion.

The *increase* of the virus is shown in such inoculable diseases as *vaccinia*, *glanders*, *malignant pustule*, *syphilis*. In all of these diseases the inoculation of the minutest portion of virus is followed by the formation of one or more vesicular structures, containing fluid from which virus, similarly and equally potent, is produced in million-fold quantity. Thus the virus of any contagious disease developed in an infected person may render his exhalations capable of similarly affecting thousands of other people. And it is probably among azotized materials chiefly that morbid poisons, whether of animal origin or of disease, find the means of their increase (CARPENTER, PAGET, SIMON).

The *transformation* of the virus is indicated by the successive phenomena which supervene during the continuous course of a specific disease. For example, syphilis is followed by a series of secondary and tertiary phenomena, which follow, on the whole, a uniform course in a great variety of patients; so that these regular syphilitic phenomena may be attributed to the *transformations* of the morbid poison; while the irregularities of the phenomena may be ascribed to constitutional peculiarities of the patient, either natural or acquired from treatment. Thus there are periods of incubation, of development, of maturity, and of degeneration in the material of the virus; and the various phenomena which constitute the *symptoms* and *prodromata* of the disease correspond to such periods of transformation; while the increasing disturbance of the general health probably implies that the morbid poison is increasing while it is being transformed—that it grows or multiplies with its development.

The *combination* of a morbid poison with some normal material of the blood is indicated by the circumstance, that when the same specific disease, produced by the inoculation of the same matter, affects many persons, the disease set up in each of them may present different peculiar features. The disease may have some peculiar and varied methods of expressing its development in different persons—"personal peculiarities," as Mr. Paget calls them, and which he considers due in some measure to the combination of the virus with one or more of those normal materials of the blood which have in each person a peculiar or personal character. By such combinations the following characters of specific diseases may be explained, namely,—(1.) Changes in the disease by transmission from one person to another; (2.) Some varieties of syphilitic sores, and varieties of their consequences in different persons inoculated from the same source; (3.) The change in the forms of secondary syphilis in transmission from parent to offspring.

The *separation* or *excretion* of the virus may be accomplished in many different ways, and may be regarded as the final purpose of

the morbid process. It is evident in the inoculable products of some vesicles and pustules.

In all of these *enthetic* diseases the immediate or the ultimate effect of the poison is to induce deterioration of the blood, and at the same time the poison seems to multiply itself, or to increase in power by some mode not quite well understood, and which has been likened to the *zymotic* action which is known to take place in fermentation. The process by which the poison is multiplied, or by which its virulence or strength is increased, varies much as to the time required for its completion.

Some of the more intense and virulent poisons, such as that of the most venomous serpents, produce their deleterious and perhaps fatal effects in as short a time as it takes the blood to complete a circulation. The change in the blood at once commences, and death rapidly follows. This sometimes happens with some forms of the *cadaveric poison*, as that which results from wounds received in the dissection of virulent cases of puerperal fever.

Other poisons do not exert their pernicious influence till after a tardy process of incubation, the time of which is not constant, as in *hydrophobia*.

In a third class of poisons which produce diseases belonging to this order, a double process of the zymotic-like action seems to take place before the full effects which the poison is capable of producing are completed. The syphilitic poison is an example of this. The multiplication of this venereal poison, and its effects upon the system, seem to become developed during the existence of the hardening process which surrounds the *infecting* venereal sore.

This is the first zymotic-like process, and is attended with a local papule, and perhaps an ulcer. From this local sore the system becomes contaminated; and in the blood a second process (of zymosis?) appears to be completed, by which the original poison becomes intensified, its pernicious influence more complete, and its specific secondary and tertiary effects are then fully developed.

Many of the diseases implanted by specific poisons claim the attention rather of the surgeon than the physician, and therefore they may be considered as not properly coming within the scope of this text-book of medicine. But for the sake of the pathological doctrines they illustrate, also because of the importance of their effects upon the system, and in relation to other diseases, some of them will be considered here.

Under the head of "Poisoned Wounds," the following *varieties* may be recognized: (a.) By venomous animals, comprehending snakes, scorpions, and stinging insects; (b.) By animals having infectious or communicable disease, comprehending *glanders*, *farcy*, *equinia mitis*, *malignant pustule*, *hydrophobia*, *cow-pox*; (c.) By dead animal matters; (d.) By morbid secretions; (e.) By vegetable substances, comprehending *arrows poisoned by wourali*, and subcutaneous injections of vegetable substances; (f.) By mineral substances.

CHAPTER VII.

DETAILED DESCRIPTION OF THE ENTHETIC ORDER OF ZYMOTIC DISEASES.

HYDROPHOBIA.

LATIN Eq., *Rabies*; FRENCH Eq., *Hydrophobie*; GERMAN Eq., *Wasserscheu*; ITALIAN Eq., *Idrofobia*.

Definition.—*A disease peculiar to animals of the canine or feline race, the specific poison of which, being implanted by them in man, or in other animals, produces a similar malady. The saliva or secretion issuing from the mouth of the diseased or rabid animal conveys the poison which inoculates rabies, either through a wound or through a thin epidermis without abrasion. The period of incubation of the poison after inoculation varies from four to sixteen weeks, or even longer, before the malady becomes developed. The disease is characterized by severe constriction about the throat, spasmodic action of the diaphragm, and distress at the epigastrium: all of which are aggravated or brought about by attempts to take fluid, or by the least breath or current of air on the surface of the body, which produces, in the first instance, an effect resembling that produced upon stepping into a cold bath. Tenacious and clammy saliva issues from the mouth. Paroxysms of phrensy, or of uncontrollable impulsive violence (rabidity), supervene. The duration of the disease varies from three to six or seven days, the greater number of cases terminating in death on the second and fourth days from the accession of symptoms. Death is generally sudden, and unexpected at the moment.*

Pathology and Symptoms.—The saliva of the dog or other animals laboring under rabies is either the virus, or contains (as any menstruum would) the poisonous principle which by inoculation produces *hydrophobia* in the human body. The disease is so named, not because there is any dread of water, but because in man the most prominent symptom is an inability to swallow, or to attempt to swallow, any fluid, on account of the extreme spasms which the attempt produces. The experiments of Hartwig have proved that the poison is of a definite character, that it may impregnate various substances, and that it retains its activity for a long period.

Two points in the pathology of *rabies* are peculiar—namely, *first*, that a long period of latency exists in the human subject; and, *second*, that inoculation is not always followed by the development of the specific disease.

With regard to the first of these peculiarities, it is to be noticed that, although in some cases pain has been felt in the cicatrix a considerable time after the accident, and in a few a slight fever or a rapid pulse has been remarked to continue from the receipt of the

injury to the outbreak of the malady, still the symptoms of the disease in man seldom show themselves sooner than the *fortieth* day after inoculation, and rarely after two years. A matured zymosis seems essential to the production of the full influence of the poison, and it may be that a double zymosis takes place, as in the case of the venereal virus, first in the part and afterwards in the system (MILLER), the result of which is either to multiply the poison or to increase its virulence.

Undoubted instances are, however, on record in which the characteristic symptoms appeared as early as the *twelfth* day (SIDEY), and on the *eighth* day (TROILLIET), who even quotes instances of their occurrence as early as the day following the injury. The duration of the period of incubation, however, is sometimes of extreme duration. It has been satisfactorily proved to extend over five and a half, six, or even nine months (BERGERON, BRANDRETH); and there is on record a large body of evidence in favor of the opinion that the incubation stage of hydrophobia may be prolonged not only over a series of months, but also of one year at least. An analysis of sixty authentic observations by Romberg has shown that the shortest interval between the introduction of the poison and the appearance of the disease is fifteen days, the longest from seven to nine months, and that the average period is from four to seven weeks. The inquiries of Drs. Hamilton and Hunter give to the majority of cases a period of incubation from *thirty* to *fifty-nine* days. In the *Transactions of the Vienna Medical Association* a case is recorded of a period of incubation extending over two years (HASSINGER); but this is discredited both by the elder and younger Gurlt, of Berlin, whose experience in veterinary pathology has been very extensive. In all such extremely long cases the question may be asked, whether the disease has been actually inoculated at a period so far back, or has there occurred a reinoculation at some intervening period? It is known that the dog in the early stage of the disease has a disposition to lick the hands, face, or other exposed parts of persons, and especially of those with whom it is familiar; and there are cases on record where the disease has been implanted in this way. Mr. Lawrence mentions the following: "A lady had a French poodle, of which she was very fond, and which she was in the habit of allowing to lick her face. She had a small pimple on her chin, of which she had rubbed off the top; and, allowing the dog to indulge in his usual caresses, he licked this pimple, of which the surface was exposed. Thus she acquired hydrophobia, of which she died." While this example teaches us that hydrophobia may be implanted without a bite being inflicted, in this almost unconscious manner, it ought to deter us from permitting such indulgences to a dog. The greatest anxiety and misery have frequently been experienced for many months by those who have been thus imprudent, owing to the circumstance of rabies having subsequently appeared in the animal so indulged (COPLAND). There are instances, however, recorded of very long periods of incubation after a bite, where subsequent inoculation, independent of a bite, could not have taken place. For example, there is a case published by Mr. Hale

Thomson in vol. i of the *Lancet*. The subject of it, a lad aged eighteen, had been twenty-five months in close confinement in prison, and during that time had never been exposed to the bite of any animal. He had been bitten severely by a dog seven years before in the right hip, and a scar still remained. During the whole period he was under observation he was sullen, gloomy, and reserved, and was never known to look the person in the face to whom he spoke. Death occurred after a three days' illness, during which "the most decided symptoms of hydrophobia were manifested." On the 15th of May, 1854, a case was admitted into Guy's Hospital, under the care of Dr. Hughes, in which hydrophobia appeared to have been developed five years after the bite (*Med. Times*, 1854).

Such observations render it extremely probable that the period of incubation of the specific poison of hydrophobia is indefinite; and the circumstances which, in man especially, seem to shorten the duration of this period or prolong it, are in a great measure quite unknown. There are some other circumstances which seem to show that during the long interval of apparent latency the quantity or the virulence of the implanted poison seems to increase, locally at least, if not also more extensively in the system.

First, In some instances there are evidences of a slow and silent change going on in the constitution, indicated by sallow looks, sunken eyes, a pulse somewhat accelerated, more easily excited and weaker, combined with symptoms of general debility (COPLAND.)

Second, The observations of Dr. Marochetti, who visited the Ukraine in 1820, and who maintained that in that country characteristic pustules were observed to form beneath the tongue, near the orifices of the submaxillary glands, between the third and ninth day after the infliction of the bite. This observation was confirmed by M. Magistel, at Boulay, in France, in 1822, who noticed that the pustules formed from the *sixth* till the *thirty-second* day. He observed two forms of pustules, a crystalline and an opaque, the latter of which, when opened, left a small ulcerated cavity. They were situated on the sides of the *frænum linguæ*, and on the lateral parts of the inferior surface of the tongue.

Third, Changes which take place in the cicatrix before the development of characteristic symptoms, indicate that the implanted poison there undergoes some process, the nature of which is as yet not known.

After the local incubation of the poison is complete, its specific action appears to be exercised upon the *medulla oblongata* and the *eighth pair of nerves*, and subsequently lesions of the structures supplied by the branches of the *eighth pair*. The action of the poison appears in the first instance to be made distinctly manifest by the œsophageal branch of the *eighth pair*, producing that derangement of function which gives rise to the characteristic symptom of the disease, or to the extreme difficulty of swallowing, especially of fluids; while the spasmodic catching of the breath, consequent even on touching the lips with any liquid, proves that the recurrent nerve is equally affected. Subsequently the eye and ear become distressed by every ray of light or impulse of sound,

and likewise the sense of touch is most painfully excited on the slightest breath of air passing over the surface of the body, all of which distinctly show that the central and spinal nerves must be functionally affected. In a still more advanced stage the suspicion, the irritability, the violence, and generally the outrageous and uncontrollable derangement of mind which often seizes the patient, bringing on epilepsy and convulsions, show that the brain itself is likewise a principal seat of the action of this terrible poison, especially the region of the *medulla oblongata*. The effects of the hydrophobic poison are often so violent in the first instance as to cause the early death of the patient; and the bodies of many persons having been examined who had so died, not a trace of inflammation or other morbid phenomena were discovered. More commonly, however, some structural alterations have been found, limited to slight inflammation of the brain, the spinal cord, or of their membranes, and of the lungs, stomach, or structures supplied by the *eighth pair* of nerves. Still, the brain, the lungs, or the stomach, may be either separately or conjointly affected—phenomena in no degree dissimilar to what have been observed in whooping-cough, where the poison seems to act chiefly on the *vagus nerve*.

The organic lesions which have been found after death in cases of hydrophobia are as follows:

When the membranes of the brain have been found diseased, the appearances have been, great congestion, especially of the *plexus choroides*, also effusion of serum, sometimes muddy, into the arachnoid cavity, and into the ventricles. In an interesting case recorded by Dr. R. W. Cunningham, of Her Majesty's 4th Bengal Europeans, the layers of the arachnoid were found adherent in many places, especially along both sides of the longitudinal sinus. The adhesions were quite soft and recent, and flakes of coagulated fibrine floated in the fluid. The brain has, in some very few cases, been supposed to be harder or softer than usual, and to have more bloody points than in health. There has been no lesion noticed, however, that could be directly connected with the malady. Changes in the *medulla oblongata* and the *spinal cord* have not yet received sufficient attention. In the case just referred to, related by Dr. Cunningham, there was a reddish spot in the substance of the *pons varolii*, having the appearance of inflammatory softening. On the lower surface of the *medulla oblongata*, at the origin of the *seventh, eighth, and ninth pair of nerves*, the membranes were highly vascular, thickened, softened, and matted together; but the substance of the nerves at their exit, and of the *medulla*, seemed normal. There are strong reasons for believing that changes actually exist in these parts which escape the detection of our unaided senses, but which the specific gravity test, combined with microscopic examination, may yet demonstrate. The mucous membrane of the pharynx and œsophagus have been seen either greatly congested or diffusely inflamed, as also that of the stomach, and of the trachea and bronchia. The latter have been found covered with a considerable quantity of frothy mucus, while the pulmonary tissue has shown marks of inflammation, though more commonly only of great

congestion. The salivary glands have likewise occasionally been observed increased in size, and vascular. In a case of hydrophobia which I had an opportunity of dissecting at Renfrew, near Glasgow, the most prominent morbid change was visible in the greatly increased vascularity of the lungs, and of the mucous membrane of the back part of the mouth, pharynx, and larynx, as far as the vocal cords. The whole of these parts were covered by a tenacious frothy mucus, tinged with blood. The glands surrounding the papillæ over the back part of the tongue were very much enlarged, not unlike what I have observed in severe cases of cholera. So also were the sub-mucous glands of the pharynx, the epiglottis, and the larynx, even in its cavity, and of those beneath the tongue. Inflammatory appearances in these parts have been observed by Morgagni, Babington, Watt, Portal, Troiliet, Copland, and others.

Symptoms.—The wound inflicted by the bite, whether neglected or dressed, generally heals up kindly, leaving a cicatrix, and for a time the patient usually suffers no other derangement of health than the depression of spirits which his apprehensions are calculated to excite. A few weeks or a few months having elapsed, the latency of the poison terminates, and the disease is formed. The course of the affection is usually divided into three stages, the first stage comprising the symptoms which precede the difficulty of swallowing; the second commences with the difficulty of swallowing, and terminates with the overthrow of the mind; the last stage embraces all the concluding phenomena.

The first stage commences in a few instances by the patient's attention being roused by a numbness extending toward the sensorium from the injured part (which, if an extremity, may become tremulous); or pain is felt in the cicatrix, sometimes severe and sometimes trifling, and which shoots up the bitten limb, following in general the course of the nerve towards the trunk. It shoots as if towards the heart, but there is no evidence of lymphatic absorption. Pain, however, is by no means constant, and is for the most part absent. In the latter case the first symptom is chilliness, with headache, or a slight attack of fever, and the patient is more excited or depressed than usual. These premonitory warnings last but a few hours, or at most a few days, when the fatal but characteristic symptom, "the difficulty and dread of swallowing"—a symptom which distinguishes this malady from all others—appears, and the hydrophobic stage commences.

The second or hydrophobic stage is ushered in with a great difficulty, if not an utter impossibility, of swallowing any liquid—a symptom which generally comes on suddenly; and such horrible sensations accompany the effort, that whatever afterwards even recalls the idea of a fluid excites violent agitation and aversion. Some patients who have been able to give some account of themselves describe the hydrophobic sensation as a rising of the stomach which obstructs the passage; others as a feeling of suffocation, or a sense of choking, which renders every attempt to pass liquids over the root of the tongue not only impossible, but which excites convulsive action in the muscles of the larynx, pharynx, and abdo-

men. In this state, says Dr. John Hunter, "the patient finds some relief from running or walking, which shows that the lungs are not yet the seat of any great oppression."

The hydrophobia, or inability to swallow fluids, is shortly accompanied by an increased flow of saliva, termed the "hydrophobic slaver." This secretion, as the disease advances, is not only copious but viscid, so that it adheres to the throat, and causes incessant spitting, and the quantity expectorated may be taken as the measure of the violence of the disease. By some this increased flow of saliva is considered as an effort of the system to eliminate the poison through these excretory glands; and therefore, mercury in large doses, to promote salivation, has been recommended to promote elimination in this way, and to reduce the extreme excitability of the nervous system (LIGGET).

The aversion to fluids is no sooner established than another series of symptoms of dreadful severity, or a highly exalted state of every corporeal sense, is added. Indeed, it is hardly possible to depict the sufferings of the patient from this cause; for not only does he shrink at the slightest breath that blows over him, but the passage of a fly, the motion of the bed-curtain, or any attempt to touch him, produces indescribable agony, almost amounting to convulsions. Dr. Elliotson states that the effect produced by these causes very much resembles that produced upon stepping into a cold bath. The sense of sight is no less a source of terror than that of touch, for the approach of a candle, the reflections from a mirror or other polished surface, occasions the same distressing effect. The hearing is as strongly affected as the other senses, so that the least noise, and especially that of pouring out fluids, throws him into a fearful paroxysm. An attendant who sat up with a hydrophobic boy made water within his hearing, which threw the sufferer into a most violent agitation. The degree to which this painful state of the senses arrives may be understood when it is stated that Magendie records the case of a deaf and dumb child who heard distinctly in this stage of the disease. The patient, thus incessantly harassed and pained by every circumstance around him, becomes peevish and irritable, and at length sees his family, relations, and strangers, with feelings of dislike and aversion, and sometimes apparently with horror.

The third stage commences by the cerebral functions becoming disturbed, the mind being either filled with dreadful apprehensions, or being so completely overthrown that paroxysms of uncontrollable impulsive violence follow. A rabid impulse overtakes the patient to tear in pieces who and whatever opposes him. This rabid impulse greatly distresses him; and it is often strongest against those to whom he is most attached, although he struggles to suppress it. In this stage horror is strongly depicted on the countenance: every symptom is aggravated, the saliva grows thick and ropy, while the poor sufferer, not daring to make the slightest attempt to swallow, spits it out incessantly, oftentimes with frequent retchings and vomiting. In this state he sometimes turns black in the face, falling into convulsions, in which he expires; or,

exhausted by his great efforts, a sudden calm ensues, and, as if nature gave up the struggle, he dies without a groan.

Remote Cause.—Hydrophobia originates in animals of the canine and feline races, as the dog, the fox, the wolf, the jackall, and the cat, as a specific inoculable disease, but from what peculiar source is altogether undetermined. It is probably at all times to a certain extent endemic, and occasionally epidemic among these animals. It has been supposed that it is excited in them by the great heat of the dog-days, or by the *æstus veneris*; but Troiliet has shown that canine madness occurs with nearly equal frequency in winter, spring, summer, and autumn. The poison is not peculiar to any country. Rabies is found equally in Europe, Asia, and America. Neither is it limited to climate. It prevails in the frozen regions of Canada, as well as in the East and West Indies. The difficulties attending any explanation of the origin of this poison are at present not to be surmounted; but hydrophobia once originated in the animals that have been mentioned, they have the power of reproducing it by their bite, not only in each other, but probably in all warm-blooded animals, certainly in all domesticated animals, as the horse, the elephant, the sheep, the ox, even in the common fowl, and in man. It will be necessary to the proper understanding of hydrophobia to give a short outline of the disease as it occurs in the dog, so constantly associated with us in domestic life, and the principal source of the disease in the human subject.

The symptoms of this formidable affection, as witnessed in the dog, are some singular departure from his ordinary habits, such as picking straws or small bits of paper off the floor, and swallowing them; licking the noses of other dogs, or other cold surfaces, such as stones or iron. Besides this, he is observed to be more lonely, shy, and irritable; his voice is so changed that his bark would not be recognized by those who have known his voice before; and he is less eager for his food, or refuses it altogether. His ears and his tail droop; his look is suspicious and haggard; and sometimes from the very commencement, there is a redness and watering of the eyes. In a short time saliva begins to flow from his mouth, he "slavers," his fauces may be seen to be inflamed, and he is feverish. The animal, though highly irritable and easily provoked, still obeys the voice of his master; and it is remarkable "that the dread of fluids, and even the sight of them—so striking a feature in man—is often wanting in dogs and other animals, for many dogs lap water during the disease" (YOUATT). In many dogs the symptoms never rise higher than these; but in others there is a repugnance to control, and a readiness to be aroused to extreme rage, on the appearance of a stick, whip, or other instrument of punishment, or on any attempt at intimidation, which strikingly characterizes the disease. In this state, however, he seldom fights a determined battle, but bites and runs away; still even this mitigated irascibility usually ends in indiscriminate aggression, till at length he dies, apparently, of convulsions or asthenia, or from mere nervous excitement and functional derangement. Magendie has inspected the hydrophobic dog, and found no characteristic morbid change. In all cases, how-

ever, in which the poison has had time to set up its specific actions, the principal lesions of structure are found to be in those parts supplied partially or entirely by the eighth pair of nerves. The tongue is swollen; the fauces, the salivary glands, and the mucous membrane at the back of the larynx behind the epiglottis, are more or less inflamed. The bronchial membrane is also occasionally inflamed, and so is the mucous membrane of the stomach, which generally contains a strange mixture of straw, hair, paper, hay, horse-dung, and earth, showing the peculiar morbid propensity of the animal; or, being void of those substances, it contains a fluid resembling the deepest-colored chocolate. Such are the symptoms and phenomena of hydrophobia in the dog, the chief source, perhaps, of this fatal malady to the human race.

The susceptibility of the human subject to this poison is by no means universal, for only ninety-four persons are known to have died out of one hundred and fifty-three bitten, making the chances of escape as three to two nearly. It has been thought this occasional immunity does not arise out of any want of susceptibility to the action of the poison, but from the person being bitten through his clothes, and the dog's tooth, consequently, having been wiped clean from all venom. Menières, however, says he met with seven cases in which the dog must have bitten through several folds, and yet they all proved fatal; showing, as he imagines, the little importance of dress as a protection from this malady.

Neither age nor sex is exempted from hydrophobia; but no instance is known of any person being affected with hydrophobia unless antecedently bitten by a rabid animal capable of communicating the disease.

It is a question of much moment whether the saliva of a patient laboring under hydrophobia will or will not communicate the disease. It may be stated as an undeniable fact that, during the many years hydrophobia has been studied, no instance is known of its having been communicated from one human being to another, although many instances have occurred of the attendants having been bitten, or otherwise accidentally inoculated with the saliva of the hydrophobic patient. The only circumstance which makes this law at all questionable is, that Magendie and Breschet inoculated two dogs with saliva taken from a diseased patient, shortly before his death from rabies, and that one dog shortly afterwards died of hydrophobia. Persons have also been seized with rabies in consequence of having wiped their lips with napkins or cloths, or other articles which were soiled with the saliva (ENAU, CHAUSSIER, and AURELIANUS).

The dog's tooth generally implants the poison, or at least some abrasion appears to be necessary, either of the cutaneous or mucous surfaces. The ancients were aware of this, for Celsus observes that the integrity of the lining membrane of the mouth is necessary to the operation of the *Psylli*, whose office it was to suck out the poison after the bite of a rabid dog; and Dioscorides expressly orders them first to wash their mouths with astringent wine, and afterwards to lubricate the cavity with oil. With regard to dogs Mey-

nill observes that "such of them as have been thought to become affected merely by the contagion of the same kennel will generally be found, upon minute examination, to exhibit the marks of bites, though concealed by the hair." When a scratch or other abrasion exists, a rabid dog merely licking the part is sufficient to implant the poison of rabies.

Diagnosis.—When hydrophobia is fully formed there is no disease with which it can be confounded; but there are many reported cases in which the imagination of a patient bitten by a dog has been so powerful as to induce symptoms resembling the disease. In hysteria the difficulty of swallowing exists, but no other symptom. Tetanus is the disease with which rabies is most apt to be confounded; yet the differences are sufficiently marked. The spasm of the muscles is more continued in tetanus; less remitting, and never intermitting. The jaw is usually much in motion in hydrophobia, in frequent attempts to clear the mouth and throat from the peculiar tenacious mucus; in tetanus it is fixed. Tetanus is rarely attended with aversion to liquids; on the contrary, the bath is grateful; nor are the tetanic paroxysms increased by the sight, hearing, or touch of fluids. Also, tetanus makes its accession usually at a much earlier period after infliction of the injury. Physiologically, while tetanus is a disease of the true spinal system, hydrophobia involves the brain also, as evinced by the disorder of intellectual function and special sense, even early in the disease. Further, the two diseases differ greatly in their mode of induction. Tetanus, in the traumatic cases, is caused by irritation of a nerve, and by disease of the spinal marrow in those which are idiopathic. Hydrophobia is the result of a specific poison introduced into the circulation, and thence affecting the nervous system as a poison would (MILLER). While in tetanus the stimulus which excites the paroxysms "operates through the true spinal cord, in hydrophobia it is often conducted from the ganglia of special sense, or even from the brain, so that the sight or sound of fluids, or even the idea of them, occasions, equally with their contact, or with that of a current of air, the most distressing convulsions" (CARPENTER).

Prognosis.—There are few instances of any patient or animal suffering from this disease having recovered.

Treatment.—As there are but very few authenticated cases of recovery from hydrophobia, so there are few instances of any mitigation of the symptoms by the use of medicine. All that remains is to mention the most leading experiments that have been made, with the hope that, as they have not been successful, they may not be wantonly repeated.

Dr. Hamilton gives twenty-one cases, and adds—"many hundreds more are on record," in which venesection has been unsuccessful, though frequent and copious. Opium has been given by Dr. Babington, to the enormous amount of 180 grains of solid opium in eleven hours, without the slightest narcotic effect, or the slightest mitigation of the symptoms. Nord has given a drachm of belladonna in twelve hours, without any benefit. Dr. Atterly gave to a child eight years old two drachms of calomel by the mouth, and

rubbed in two ounces and a half of strong mercurial ointment in a few hours, with an equal want of success. A case, however, is related by Ligget, which is said to have been successfully treated by half-drachm doses of calomel, given to the extent of ptyalism, induced in three days, after four and a half drachms of calomel had been taken. The case really appears to have been one of hydrophobia; and recovery is said to have been complete by the twelfth day (*Amer. Quar. Journal of Med. Science*, Jan., 1860). Iron, arsenic, nitrate of silver, camphor, musk, cantharides, turpentine, tobacco, acetate of lead, ammoniacal solutions of copper, hydrocyanic acid, galvanism, strychnine, nitrous oxide, chlorine, and guaiacum, have all been given in equally large doses, but have signally failed. These include some of the most powerful medicines in the Pharmacopœia; and, in addition to these, Ploucquet, in his *Literatura Medica Digesta*, has enumerated nearly 150 others.

The failure of every remedy by the mouth, and the inefficacy of opium, of morphine, and of laurel-water, even when injected into the veins, so convinced Magendie that in hydrophobia the constitution was armed against the action of any medicinal substance, that on a patient laboring under this disease being brought to the Hôtel Dieu, he determined to rely for all treatment on an injection of warm water into the veins. The patient, at the time of the operation, is represented as being absolutely insane, so as to require to be restrained. In this state, and with a pulse of 150, Magendie injected into his veins, in the course of two hours and a quarter, two pints of water, at the temperature of 100°. At the conclusion of this operation the pulse had fallen to 80, and the patient recovered his senses, so that restraint was no longer necessary. The sequel, however, renders it doubtful whether this mitigation was desirable, at the price of the intense suffering which followed. The poor man lived eight days afterwards, but the despondency and mental agitation quickly returned, and at the end of three days the poison (or the state of the blood induced by it and the warm water) appeared to set up a new series of actions on the synovial membranes of the wrists, elbows, and knees, attended with excessive pain, so that he was unable to bear the weight of the bed-clothes, and he died in great torture. The articulations thus affected were found, on post-mortem examination, to be greatly inflamed, and their cavities filled with pus. This case is remarkable as being the one in which life was prolonged for the greatest period of time recorded of this disease. The experiment has since been repeated by Gaspard and others; but the mitigation, if any, has been so slight and transient as to give no encouragement for repeating it; and, tried on the rabid dog by Youatt and Mayo, it proved eminently unsuccessful.

The property which some animal poisons have of controlling and of interrupting the actions of other morbid poisons on the constitution has caused even animal poisons to be tried in the cure of this disease. The rapid and powerfully acting poison of the viper led to the hope that the bite of that reptile might prove an antidote to the hydrophobic virus; but the experiment, tried in France, Ger-

many, and Italy, upon animals, has been entirely unsuccessful. M. Grindard conceived that the vaccine virus might influence hydrophobia, and he vaccinated a hydrophobic child in three places, and afterwards injected five charges of vaccine lymph into the veins; but the child died without any marked remission, and in the usual time. The following draught has been found rather to promote euthanasia than to hold out any prospect of cure:

B. Spirit. Æther. Sulph., Tinct. Opii, āā ℥xx; Spirit Ammon. Aromat., ℥ss.; Chloroform, ℥xx; Mist. Camph., ℥iss.; *misce.* To be given as often as may be considered safe (CUNNINGHAM, CARDEN).

On the same principle chlorodyne ought to be given. (For its composition, see page 454, *ante.*) The vapor bath is sometimes useful in moderating spasm.

Preventive Treatment.—The probabilities are, that unless the operation of excision, or cauterization, be performed within a few minutes after the bite of the rabid animal, it is impossible to save the patient from the fatal disease, which, according to the susceptibility of his constitution, may threaten him at any moment. In all probability no prophylactic medicine exists in nature, and the administration of any potent substance by way of prevention is worse than useless; for, without protecting the patient, it may injure his constitution. Mild remedies, if they tend to tranquillize his mind and appease his apprehensions, may be innocently employed.

The theory which maintains that a zymotic incubation first takes place in the wound, by which the poison is originally implanted, suggests the most rational prophylactic—namely, to destroy entirely by *potassa fusa* the whole cicatrix, where practicable, or by some other surgical means entirely to remove it, at as early a period as possible, and *previous* to the occurrence of symptoms. When premonitory symptoms are first observed, the following plan has the recommendation of Dr. Maxwell in *The Indian Journal of Medical and Physical Science*, and of Dr. Copland, namely,—(1.) That the original cicatrix be freely laid open, and suppuration from it speedily and freely produced. (2.) The nerves, or nerve, leading to the part are to be divided without delay, the more remote from the wound the better. (3.) Free perspiration should be promoted by the hot air bath. (4.) Bleeding from the arm to syncope in robust persons with sthenic symptoms, or cuttings on the nape of the neck, are modes of practice indicated by the lesions found after death.

GLANDERS.

LATIN EQ., *Equinia*; FRENCH EQ., *Morve*; GERMAN EQ., *Rotz*; ITALIAN EQ., *Cimurro*.

Definition.—*A febrile disease of a malignant type, resulting from the implanting of a specific poison from glandered horses. It is characterized by vascular injection of the nasal mucous membrane, from which*

an aqueous, viscid, glutinous, or purulent discharge proceeds, on which chancre-like sores are formed, extending to the frontal sinus and neighboring mucous surfaces. The lymphatic glands enlarge in the vicinity of these mucous membranes. A tubercular or pustular eruption appears upon the skin, followed by suppurating, bloody, or gangrenous ulceration in various parts. A general inflammation of the lymphatics and of the glands may occur, giving rise to the small tumors known as "farcy buds" or "farcy buttons." These gradually suppurate, and secrete a specific virus.

Pathology.—The horse, the ass, and the mule are liable to a disease termed glanders. It occurs under two forms, named respectively *glanders* and *farcy*, being "an inflammatory affection of the skin and of the absorbent system, produced by the contagion of matter from a horse having glanders or farcy." Many veterinarians have considered these varieties to be distinct diseases; but numerous experiments have demonstrated that they have their origin in the common animal poison. It appears, however, that there are several grades or varieties of both these diseases. Thus, if glanders be defined to be a fever with a running of matter from the nose, farriers distinguish three kinds: one consists of swelling, ecchymosis, and gangrene of the mucous membrane, with a discharge principally from the pituitary, tracheal, or bronchial membrane; another, of a pustular eruption of the same parts, followed by ulceration; while a third consists in a combination of these two forms of disease. Of farcy, also, there are two kinds: the *bud farcy* and the *button farcy*. The "bud farcy" consists in the formation of a number of tumors on different parts of the body, as on the head, neck, and extremities, and particularly on the hinder ones, these tumors being formed not only by enlargement and inflammation of the glands, but also of the areolar tissue, and which, at the end of four or five days, soften and ulcerate. Similar tumors are said to form in the substance of the pituitary membrane, which quickly suppurate and cause death. The "button farcy" is an inflammation limited to the lymphatic glands and vessels, without involving, in any considerable degree, the areolar tissue. It usually commences in the hinder extremities, causing lameness and enlargement of the limb: and when the valves of the lymphatics become thickened, it forms a tumor called the "farcy bud;" while if the lymphatic vessel itself be inflamed, it is termed "farcy pipe." It may be shortly stated, that in *glanders* the nasal passages especially suffer; while in *farcy* it is the lymphatic system which is affected.

The mildest form of all is that named "*Equinia mitis*," and thus defined: "*A pustular eruption, produced by the contagion of matter from a horse affected with 'the grease.'*"

It has been determined by a number of severe accidents occurring to persons employed about glandered horses, that the poison producing them is capable of being transmitted from the horse to the human subject, and again from the human subject to the horse and to the ass; and there is reason also to believe that it is capable of being transmitted from one human being to another (Zimmerman, in *Virch. Arch.*, vol. xxii., p. 209, and *Year-Book of New Syden.*

Society, 1862). The attention of the profession was first called to this interesting subject by Mr. Muscroft, in *The Edinburgh Medical and Surgical Journal*, in the year 1821, where he relates the case of the whipper-in of the Bradworth hunt, who wounded himself in cutting up a glandered horse for the kennel, and died, at the end of a week, of confirmed glanders; and two similar cases appeared in the same work about two years afterwards. Simultaneously with Mr. Muscroft, Dr. Copland, in the course of a discussion at the Medico-Chirurgical Society of London, stated that the fact of the disease having been thus communicated had been proved by cases that had occurred in Germany, and which were published in *Rust's Magazine* for 1821. The cases excited but little notice till Mr. Travers published his valuable work on *Constitutional Irritation*, in 1828, containing a letter from Professor Coleman on the transmission of glanders from the horse to man, and from man to the ass, together with some other cases which had fallen under his own observation. The subject was now followed up by Dr. Elliotson, in two papers in the *Transactions of the Medico-Chirurgical Society*, narrating three cases which had occurred in his own, Dr. Roots's, and Dr. Williams's practice. At length all then known facts were collected in an elaborate paper by Rayer, in the sixth volume of the *Mémoires de l'Académie Royale de Médecine*.

In the cases collected by Rayer, the nose and nasal fossæ had only been examined in four cases out of fifteen, and in these there was found either ecchymosis, ulceration, or gangrene of the mucous membrane of the *septum nasi*, or of the sinuses. The mucous membrane of the larynx, or trachea, has likewise been found studded either with the peculiar eruption, or diffusely inflamed or ulcerated, so much so that in one case the epiglottis was in part destroyed. The lungs have likewise been found either gorged with blood, or the seat of lobular pneumonia, or of vomice, with typhoid symptoms—*broncho-pneumo-typhus*, as it is called in Germany. In Dr. Roots's case there was an encysted abscess of the lung, which contained about two ounces of pus. Besides these affections of the more vital organs, a number of small farcy tumors have been found in different parts of the trunk and extremities, and perfectly remote from the point originally punctured. These tumors were in different states of inflammation, some being white and indurated, others soft and injected, and others in a state of suppuration. In Dr. Roots's case an abscess on the back of the hand communicated with the articulation of the metacarpal bones; and in another case an abscess had opened into the knee-joint. The absorbent vessels have likewise been found inflamed along the arm from the point of puncture, or site of primary inoculation, and the glands to which they lead have been found enlarged and indurated, or in a state of suppuration.

The result of all these observations shows that in cases of glanders a specific poison is implanted which infects the blood, and, after a given period of latency, produces, in slight cases, an abscess at the point of puncture, followed by some tumors in the course of the absorbents connected with the punctured part. In severe cases

fever is previously set up, and after this has continued for some days, there follows either a diffuse or an eruptive inflammation of the mucous membrane of the nostrils and of the trachea, terminating in suppuration, ulceration, or gangrene; also some inflammatory affection of the lung, together with the usual farcy button or bad tumors in different parts of the body.

Symptoms.—Glanders may be either acute or chronic. Acute glanders is expressed by primary fever, followed by local inflammation; chronic glanders, when the local inflammations exist *per se*.

The acute disease is ushered in by an attack of primary fever, with or without rigors, and followed by pains in the limbs so severe as often to be mistaken for an attack of acute rheumatism.

Some days after, the pained parts become the seat of phlegmonous tumors, accompanied with much pain, redness, and tenderness; these more commonly terminate in abscess, sometimes discharging a laudable pus, but more usually a bloody sanies, and rapidly become gangrenous. Towards the close of the disease, in almost all cases there has been a discharge of matter more or less purulent, viscid, and mixed with blood, from the nostrils. The quantity, however, has in general been inconsiderable, and sometimes scarcely appreciable. The period at which this symptom appears is not constant. It has been seen as early as the fourth, and as late as the sixteenth day. In the course of the disease the eyelids are generally tumefied, and discharge a thick viscid matter, like that from the nose; and enlargement of the submaxillary glands occurs.

One of the most remarkable symptoms of acute glanders in man is the eruption of pustules on the face, trunk, limbs, and genital organs. This eruption has been compared to *varicella*, to *small-pox*, and to *ecthyma*; but in fact it is an eruption *sui generis*, and cannot be compared to any other. It has been observed to occur about the twelfth day, and to be preceded and accompanied by profuse fetid sweats. Besides this eruption, a number of black bullæ have been observed on the nose, forehead, below the ears, on the fingers, toes, and genital organs, and these have been followed by gangrene more or less extensive and deep.

The pulse is full and quick in the early stages, but towards the close it becomes rapid, small, irregular, and even intermittent. The tongue varies, as in typhus, being first white and coated, and subsequently brown or black. Diarrhœa and meteorism often complicate the disease, and blood has been observed in the stools. Cerebral disturbance has come on as early as the second day, but more commonly not till towards the tenth; sometimes marked by a singular want of intelligence, at others by a sinister presentiment, followed by stupor and death.

Acute glanders is rapid in its course, and two-thirds of the cases have terminated before the seventeenth day; some have died on the twenty-first day, a few on the twenty-eighth day, and only one has survived till the fifty-ninth day.

Chronic glanders, or farcy, differs from acute glanders in the circumstance of the local lesion preceding the general febrile derangement, the introduction of the poison being followed in a few hours

by inflammation of the lymphatics proceeding from the wounded part, and extending sometimes to the elbow or axilla, and involving the axillary glands. The effects are followed by inflammation and extensive abscesses in the subcutaneous cellular tissue, often involving the whole limb. From this state the patient may recover; but should these abscesses be multiplied over various parts of the body, and be accompanied either by the pustular or gangrenous vesicular eruptions, or by both, the result is generally fatal; hectic symptoms supervene, and hasten the final catastrophe.

The disease has terminated within a fortnight, but more commonly it has not proved fatal till the end of a month; and, in cases still more chronic, a twelvemonth has been known to elapse before the patient finally recovered or died. Such are the general phenomena of acute and chronic glanders, as they have been observed in the human subject.

Cause.—The remote cause of glanders in the horse is but little understood. It is probably due to a specific miasmatic poison, having a peculiar affinity for the horse, and animals of his class. Glanders, however, when it affects the human subject, has in all instances been distinctly traced to the glandered horse as the remote cause. No instance is known of the disease occurring primary in man.

In the horse certain predisposing causes greatly favor, and are perhaps necessary to, the spread of glanders, such as dirty, close, ill-ventilated stables, especially if the situation be low and damp. Horses when crowded on board transports are greatly liable to this affection. The Arab, in transporting his horses from Arabia to India, always chooses that part of the year when the passage is shortest, lest the accidents incident to a long voyage might oblige the hatches to be closed, and want of ventilation promote the development of glanders. Bad food is a powerful predisposing cause in the horse, especially when these animals are picketed on service, and thus exposed to the inclemency of the weather. At the close of a campaign the cavalry are often decimated by this disease, and towards the termination of the Peninsular war the losses from this cause are said to have been enormous. The cases occurring in the human subject are too few to allow of any inference being drawn as to the influence of the predisposing causes in the production of glanders; but the disease generally occurs in young men; and probably a close investigation would have shown that the habits of the patient were such as to fall within those laws which favor the production of the disease in the horse.

The majority of veterinary surgeons, of stable-keepers, and coach proprietors, believe that the disease is contagious among horses, and if a glandered horse has been introduced into stables, the stock in these stables have become diseased. There are few districts in which some farmer, by the loss of a considerable part of his team, has not had sufficient proof of the communicable nature of glanders. In this country the law is severe against offering for sale, or even working a glandered horse; which shows that the opinion of our ancestors, time out of mind, has been that glanders is a contagious and a fatal

disease. In Germany the belief of contagion is so general that it is said the law directs any horse that has been in contact with a glandered animal to be immediately killed. Again, Professor Coleman has produced glanders by direct inoculation from horse to horse; so also have Professors Peal and Renault; while Leblanc assures us that he has repeated these experiments till he has demonstrated that not only is glanders contagious, but that farcy and glanders are mere varieties of the same disease,—the farcy matter producing glanders, and the matter of glanders producing farcy.

Cases of the transmission of glanders from the horse to man are now numerous; and that the disease is actually glanders has been shown by Professor Coleman, who directed two asses to be inoculated with matter taken from the arm of a person then laboring under this disease, consequent on a puncture received in dissecting a glandered animal, and both animals died of glanders. These experiments have been repeated, with similar results, by Gerard, Hering, of Stuttgart, and more recently by Leblanc, with matter taken from a patient that died glandered under the care of Rayer, so that no doubt can exist of the fact. It seems proved, therefore, that glanders is transmissible from the horse to man, and again from man to the ass. It has been contended, also, that if glanders is transmissible from man to animals, the disease must be capable of being communicated from one human subject to another; and a case of this description appears actually to have occurred in St. Bartholomew's Hospital about twenty years ago, when the nurse, a healthy woman, contracted the disease from a patient in the ward, and, after a short illness, died with every symptom of glanders.

The fact of repeated inoculation with glandered virus distinctly shows that fomites may be so infected as to produce the disease. The spread of the malady has been attributed to healthy horses having drunk out of the same pail or trough with a glandered horse, or to licking the neighboring rack or partitions of the stalls in which a glandered horse has been placed. Mr. White attributes the occurrence of glanders in a mare and two foals to some hay left by a team of glandered horses being blown into their paddock.

The specific poison of glanders has been introduced into the system both by the cutaneous and mucous tissues. The disease has been produced by inserting the virus under the cutis with a lancet, and by rubbing it on the greasy heel of a horse; it has also been produced by inoculating the mucous membrane of the nose of the horse, or by smearing that membrane with farcied matter. Farcied matter has also been made up into balls, and introduced into the stomach of the horse, and glanders has resulted. There can be no doubt, therefore, that the poison is absorbed both by the cutaneous and mucous tissues, and that, being absorbed, it infects the blood. This latter fact has been distinctly proved by Professor Coleman. "I have," says this gentleman, "produced the disease by first removing the healthy blood from an ass, until the animal was nearly exhausted, and then transferring from a glandered horse blood from the carotid artery into the jugular vein of the ass. The disease in the ass was rapid and violent in degree; and from this animal, by

inoculation, I afterwards produced both glanders and farcy. In acute glanders, therefore, the blood is undoubtedly affected."

Period of Latency.—The poison of glanders has its period of latency, like all other morbid poisons, and that period is in general short. Two asses were inoculated by Mr. Turner, the one about a year and the other a year and a half old, and in the first the maxillary glands became tender on the second day, and the discharge from the nostrils was established on the third. In the other the maxillary gland enlarged on the third day, but the discharge from the nostrils did not take place till the sixth day. Sometimes, however, the incubation is much longer. In the *Procès-verbal de l'École de Lyon* a case is given of a horse which was inoculated with farcy matter, but the disease did not appear till the end of three months, and then precisely at the points of puncture. M. Gerard, an ex-veterinary surgeon of the French "artillerie de la garde," states that he introduced the matter of the discharge every day into the nostrils of certain horses, by means of a brush, and that the disease appeared in one on the seventh day, but in two others not till the thirty-second day.

In the human subject the poison has in general been latent from two to eight days after the accident of inoculation.

Prognosis.—Of fifteen cases of acute glanders collected by Rayer only one recovered. Of fifteen cases of acute farcy only five recovered. Of seven cases of chronic farcy only one died. Of the three cases of chronic glanders two died. A favorable prognosis consequently, is only warranted in the chronic form of the disease.

Diagnosis.—"Acute glanders," says Rayer, "cannot be confounded with poisoning from puncture in dissecting or opening dead bodies; for," he adds, "out of fifty such cases reported by various authors, no mention is made in them of a discharge from the nostrils, or of a nasal or laryngeal eruption being found after death, or of the peculiar cutaneous eruption." Leblanc also states that he has inoculated the horse with a great number of other morbid secretions from the human subject, but has in no instance produced any disease similar to glanders. It may for a short time be mistaken for rheumatism, but the occurrence of the secondary actions quickly dispels this error.

Treatment.—All the remedies hitherto tried in acute glanders have failed. The coming on of typhoid symptoms has led to the administration of *quinia*, *valerian*, *serpentaria*, *ammonia*, and other stimulating medicines; but all of them have failed. Vomiting and purging have likewise been had recourse to; but these measures have been equally unsuccessful. It is probable, therefore, that the cure of this disease depends on the discovery of a specific remedy, and experiments in treatment may be warranted as the only chance of subduing a malady which has so constantly proved fatal. In the more chronic forms of the disease the recovery of the patient has appeared to be owing to the excellence of his constitution during the natural elimination of the poison, to good ventilation, and to generous diet, rather than to any powerful effect produced either by general or local treatment.

Preventive Treatment.—The prophylactic treatment is the same as that of all other contagious diseases—namely, being careful to avoid all contact with the morbid poison, and especially when a finger or other part of the hand is abraded; and if by accident the veterinary surgeon should inoculate himself, he ought instantly to destroy the part with *potassa fusa*. It has been recommended, after the disease is set up, to extirpate the enlarged glands; but according to the doctrines set forth in the text, this practice is as unwarrantable as hopeless.

MALIGNANT PUSTULE (VESICLE?)

LATIN Eq., *Pustula pestifera*; FRENCH Eq., *Pustule maligne*; GERMAN Eq., *Milzbrand*—Syn., *Karbunkel-krankheit*; ITALIAN, *Pustula maligna*.

Definition.—The result of a specific poison implanted on some uncovered part, which produces, in the first instance, a redness like the bite of a gnat, and afterwards a minute vesicle. A peculiar form of spreading gangrenous inflammation is excited, which rapidly spreads from the point first affected to the neighboring tissues. Hardening and blackening of this part is so extreme, and death of tissue is so entire, that the part creaks when cut with a knife—no pain attends the incisions, crops of secondary vesicles form round an erysipelatous-like areola, chains of lymphatics become inflamed, the breath fetid, and death follows amid all the indications of septic poisoning (BUDD).

Pathology and Historical Notice.—This disease has been long familiarly known and described by French, German, Russian, Swedish, Lapland, and Italian medical men; and it proves fatal every year to a large number of persons in various parts of Europe. British medical men are not generally familiar with the disease; and its occurrence in this country escaped general recognition till the admirable papers of Dr. William Budd on the subject (read at the meeting of the Medical Association in London, and published in the *British Medical Journal* of 1863) gave a full account of the literature of the subject, and showed that malignant pustule has been long known in this country as an epizootic, causing every year a large mortality among English live stock. The “joint murrain,” “black quarter,” or “quarter evil,” and the “blood” (the name by which the malady is known in the sheep), are the same diseases as the “charbon,” “quartier,” and “sang” of the French, and the “milzbrand” of the Germans. From the writings of Dr. Budd on this subject the following account of this remarkable and terrible disease is taken. The disease has prevailed from time immemorial, in various continental countries, in oxen, sheep, horses, and other animals; and, concurrently with the cases of malignant pustule, which are the result of direct inoculation from the morbid material of those animals, other cases occur in which the exact vehicle of the poison cannot be identified; but these cases have all the significant peculiarity, that the disease is always seated on some part of the person which is habitually uncovered.

In animals, and especially in oxen, the action of the specific

poison seems to be even more virulent than it is in man. Death is more speedy; there is a more rapid spread of gangrene; and while the animal is yet living, the extrication of fetid gases from the tissues of the parts affected goes on to a great extent. The contagious property of the poison is possessed in the highest degree by the lymph contained in the characteristic vesicles, and, next to this, by that peculiar exudation which occurs in the areolar tissue of the affected part, and in that of various parenchymatous organs, and sometimes in the serous cavities of the chest and abdomen. The identity of the malignant pustule of man with the "*charbon*" of cattle has been satisfactorily proved by the fact that the disease when contracted by man, has been communicated back to the animal by inoculation from man.

[Inoculation of the human being with matter from a braxy sheep will produce malignant pustule, and the inoculation of the matter of malignant pustule of man in a sheep will produce braxy (RENAULT).]

It is only at the onset that the disease is a local one; but very soon general poisoning ensues, which is due to the after-diffusion of the morbid changes and products engendered in the part first affected. This is a very important point in the pathology of the disease, and with a view to successful treatment; for the early destruction of the diseased part by caustic not only prevents the development of the constitutional disorder, but in many cases issues in a perfect and speedy cure.

Propagation.—The disease may be communicated to man in the following ways: (1.) By direct inoculation, as in the case of butchers, farmers, skinners, herdsmen, drovers, and others, in whom accidental inoculation with it appears to be an event of no uncommon occurrence in countries where "*charbon*" is most rife. (2.) By means of the skin, or simply by the hair of diseased beasts. Trousseau, for example, relates that in two factories for working up horse hair, imported from Buenos Ayres, and in which only six or eight hands were employed, twenty persons died in the course of ten years from malignant pustule. There are many other cases related by Dr. Budd, and some which clearly show that the virus of *malignant pustule*, like other contagious poisons, when once in the dried state, may retain its powers for an indefinite period of time. The disease may thus be propagated through contact with bones, hoofs, horns, and the fat and tallow of animals dead of the "*charbon*." (3.) The disease may be communicated by eating the flesh of animals killed while affected with it, as also by using the milk and butter of affected cows.

[There is a good deal of diversity of opinion and discrepancy of evidence with regard to the risk of eating the flesh of animals affected with malignant pustule. Ramazzini, Lancisi, Caillot, Enaux, Chaussier, Foderé, Gamgee, Rendle, and others, give many instances where the disease has been developed in such as ate of the meat of cattle suffering from carbunculous fever. Dr. Livingstone (*Travels in Southern Africa*), says, that those who eat the flesh of animals who die from pleuro-pneumonia,

are attacked with carbuncles. Menschel (quoted by Parkes, *Manual of Practical Hygiene*, 2d ed., p. 173), states that twenty-four persons were attacked with malignant pustule, the majority after eating the flesh of beasts suffering from the disease, the others from direct inoculation; five died. Dr. Samuel R. Percy (*New York Medical Journal*, August, 1866), mentions, that on the third day after eating sparingly twice of a piece of beef which had "a peculiar swilly odor and taste," he was taken suddenly sick while in the street, with severe pain in his left shin. On his return home, he found "an inflamed spot, about three inches in diameter, and in the centre of this, two pustular elevations, each about the color and size of a split pea. The next day the inflamed surface had become a vesicle; eventually the whole skin peeled off, and the two pustular spots were deep-seated ulcers; six weeks elapsed before they were perfectly healed." On the other hand, Parent Duchâtelet, Levy, Pappenheim, Morand, Duhamel, Thomassin, and Neffel, relate cases where the flesh of animals with malignant pustule have been eaten with impunity, and that too in several instances where the disorder had been communicated by inoculation; and Thudichum (*Brit. Med. Jour.*, April 28, 1866), maintains that there is no satisfactory evidence to warrant the belief that man can be infected by eating the meat of animals so diseased.]

(4.) Insects which have been in contact with the bodies or carcasses of diseased cattle may communicate the disease to man. Most commonly it is the insects with piercing probosces, such as gad-flies, after having sucked the putrid juices of dead or sick animals, and then settled on the persons of men, which effect the inoculation; but flies which make no wound may also implant the poison on the skin by their soiled wings and feet (VIRCHOW, BOURGEOIS). The latter observer says "he has seen the disease produced by the puncture of a gad-fly which came out of a fleece of wool" (BUDD).

[Although, in nearly every instance, malignant pustule in man is communicated directly by the virus of an infected animal, still there are a few exceptional cases where, it would seem probable, it was of spontaneous origin, under certain special conditions (*Recueil de Mém. de Méd. et de Chir. Mil.*, t. 1859; MANOURY, FOURNIER, BAYLE, MARET, SWYGENHOVEN).]

Phenomena and Symptoms.—A considerable degree of pruritus in the part is succeeded by the appearance of a red spot like a flea-bite. A vesicle, in the course of twelve or fifteen hours afterwards, may be observed, at first about the size of a millet-seed, but very soon it acquires larger dimensions, and, if not ruptured by the patient, bursts spontaneously, and dries up in about thirty-six hours, leaving the exposed *cutis vera* dry, and of a livid color. Twenty-four or thirty-six hours after the attack (itching having now ceased), a small, hard, and circumscribed nucleus—the "*parent nucleus*" of Virchow, the "*maltka*" of the Russians—having the form and size of a lentil, is perceptible under and around the seat of the vesicle. In the circumference of this a soft but still resisting swelling, of a reddish or liver color, forms an inflamed areola, and becomes covered eventually with secondary sero-sanguinolent vesicles, similar to the vesicle

which first appeared. These are at first isolated, but speedily they become confluent. The central spot may contain at first a transparent, bright yellowish fluid, which very early becomes reddish or bluish; then of a brownish hue, when the spot becomes extremely hard, very insensible, and rapidly becomes gangrenous. The inflammation extends to a considerable distance, both in depth and circumference; the neighboring skin is red and shining; the subcutaneous areolar tissue is puffy and emphysematous-like; the excoriated surface readily dries up, and becomes, as it were, mummified; and in its neighborhood new vesicles spring up, which run the same course as the former. The part soon loses its vitality, so that it may be pierced with needles without the patient becoming aware of it. It is also a remarkable feature of *malignant pustule*, that severe pain is generally absent. If the disease ceases to advance, an inflamed circle of vivid redness now surrounds the gangrenous portion, the tumefaction diminishes, and the patient experiences something like an agreeable warmth, accompanied by a pulsatory motion of the affected part. The pulse, which before was irritable and feeble, begins to revive, strength increases, a gentle perspiration indicates the crisis of the febrile state, and nausea ceases. Separation commences between the living and the dead parts, and is attended by copious suppuration. If the disease should not tend to a favorable issue, suppuration does not take place; the gangrene spreads rapidly; the pulse becomes smaller and more contracted; the patient suffers from extreme lassitude and inability to sleep; and, finally, with a tendency to syncope, he becomes passive as to the result. The tongue is dry and brown, the features shrink, the skin is parched, the eyes are glassy; and cardialgia and low delirium indicate the approach of the fatal termination (BUDD, RAJER, VIRCHOW, BELL, CRAIGIE).

The face (often in the lip, or immediate neighborhood of the mouth), the neck, the hands, the arms, and the legs, are almost the only parts on which it appears; and if by chance it becomes developed on other parts, we may be sure the poison has been carried there directly by the fingers, or other agents impregnated with the virus. The phenomena, therefore, which such cases exhibit in man are identical in every particular with those which have been seen in farriers, and others in continental countries who have the charge of cattle, and who in numberless instances are known to have become diseased from the accidental but direct inoculation of the "*charbon*" virus.

[*Malignant œdema of the eyelids* would seem to be identical in its nature and origin with malignant pustule (DEBROU,* BOURGEOIS,† RAIMBERT‡). It begins with itching, quickly followed by great swelling, so that very soon after the onset the lids cannot be forcibly separated, owing to the degree of serous infiltration. The skin is tense and smooth, and

* [L'Œdème Malin ou Charbonneux des Paupières. Par le Dr. Debrou. Gazette des Hôpitaux, No. 183, 1860. Arch. Gén. de Méd., Oct., 1865.]

† Traité de la Pustule Maligne. Par Bourgeois (d'Etampes).

‡ Traité des Maladies Charbonneuses. Par L. A. Raimbert. Paris, 1859.]

without change of color. No excoriation, vesicle, pimple, or areola, no "parent nucleus," can be seen. The swelling soon extends to the temple, forehead, and cheek of the affected side, subsequently invading the lips, nose, chin, and neck. It is hard, pale, and indolent. There is neither headache nor nausea, and the pulse is regular. If the part has been early cauterized, there is a continuous weeping of a yellowish serosity. Unless the disease is arrested within the first day or two, constitutional infection is announced by a chill, or a sensation of general coldness; the pulse becomes quick and feeble, with excessive prostration; the respiration is embarrassed; there are nausea, vomiting, jactitation, delirium, and cold extremities, ending, in from two days to a week from the initial symptom, in death.

Anatomical Characters.—An examination under the microscope, by M. Robin, of a malignant pustule excised by M. Manoury, showed nothing peculiar in its structure. There was a granular appearance, analogous to what is seen in all gangrenous tissues. The meshes of the subcutaneous connective tissue are enlarged, and its fibres are of a brownish color, due to serous infiltration (GOUJOT). The absence of the inflammatory process in the oedematous tumor is an important and essential character of malignant pustule, which seems to oppose the formation of pus, so long as the infecting virus is present (BOURGEOIS, SALMON, MANOURY). The blood is fluid, fetid, and gaseous; the corpuscles broken down, and vibrios are found in it, which, according to Duvaine, are bacteria (LEURET, HAMONT, BRAUVELL). The heart is softened; and the spleen reduced to a pulp, and contains bacteria. In the stomach and small intestines there are a number of round, elevated patches, resembling, when scraped off, Indian ink, or the vomit in cancer of the stomach. A vertical section shows them to be limited to the mucous membrane—which is sound immediately around them—and composed of a homogeneous, almost black substance, which examined microscopically is found to be altered blood, with a large number of amorphous granules of hematoidin (DAVAINE). Hence they are not, as stated by some writers, internal malignant pustules, or gangrenous eschars.

As far back as 1850 Davaine found in the blood of braxy sheep certain vibrios, which resembled bacteria in all respects except being without movement. To these he gave the name of *bacteridia*. Since 1863 he has made a number of experiments, with the view of elucidating the question, and ascertaining what actual relation there was between the presence of these bodies and charbon virus, and with the following results: (a) *Bacteridia* are found in every disease of this nature (*dans toute maladie charbonneuse*), whatever its form, and in every animal affected with the disease. (b) The presence of these vibrios in the spleen, liver, and blood, precedes the development of the morbid phenomena. (c) The blood ceases to be infecting when *bacteridia* can no longer be found in it (*Archives Générales de Médecine*, t. i, 1868).]

Treatment.—The affection only admits of cure when to the uninforming aspect is trivial; and it can only be cured by a process which leaves a mark. To make an abiding scar on the face, for the treatment of what, at the worst, appears to be no more than a common boil, is a serious consideration for the reputation of the medical man in his practice amongst ignorant people.

The progress of the disease is only certainly to be averted by the use of caustics; and of the various caustics in use the evidence ap-

pears to preponderate in favor of *potassa fusa*; although Chaussier and others prefer *nitric acid* or the *chloride of antimony*. But everything hangs on the recognition of the disease in its first stage (BUDD).

[M. Manoury excises the pustule, and covers the wound with corrosive sublimate in powder. MM. Mauvezins cut out the "parent nucleus," or the entire vesicle, and then use the actual cautery at a red heat, and claim that, when resorted to at once, it hinders general toxic symptoms, and invariably cures (*Arch. Gén. de Méd.*, March, 1864, and June, 1866). Bourgeois relies on caustic potash, which he applies, removing the eschars, until blood appears. Most of the French surgeons depend on the actual cautery. It is probable that chromic acid, from its rapid and powerful action, would prove efficient, followed, after wiping away the detritus, by the free application of a strong solution of the chloride of zinc, 50 grs. to the ounce of water. As soon as the constitutional symptoms set in, alcoholic stimulants and carbonate of ammonia should be largely given, together with concentrated nourishment.]

SYPHILIS.

LATIN Eq., *Syphilis*; FRENCH Eq., *Syphilis*; GERMAN Eq., *Syphilis*; ITALIAN Eq., *Sifilide*.

Definition.—*The result of a specific poison implanted on some part of the body, but generally through an abrasion or sore consequent on sexual intercourse with an infected person. A peculiar series of phenomena supervene, which mark the general infection of the system. The principal anatomical signs of general infection consist of induration (specific) round the spot where the virus has been implanted, induration of the lymphatic system of glands, the formation of nodes or gummatous nodular tumors in the connective tissue generally, and especially in that of the true skin, bones, mucous membranes, and solid visceral organs—e. g., liver, brain, lungs, and heart. A cachectic condition of the system accompanies the phenomena of infection; and indurations may remain in the form of hardened fibrous tissue in various parts of the body for an indefinite period of time.*

Pathology and Morbid Anatomy.—Advances in Pathology of late years have not been more marked in any direction than in demonstrating the very remote effects which syphilitic infection exercises upon the organs and the constitution of man. These advances are due to clinical, experimental, and post-mortem, observations. They have shown that a considerable number of doubtful cases of ill-health are in reality due to the specific poison of that venereal disease to which the name of *syphilis* is now restricted, whose morbid effects are not fully developed till many days, months, and even years after inoculation. Hitherto surgeons have claimed the subject of *syphilis* as their peculiar field; but after the surgeon had healed the sore, the morbid influence of the poison in many cases still remained, and internal lesions, impaired health, and degenerate constitution, eventually brought the patient to consult the physician as well as the surgeon.

The pure surgeon and the pure physician must, therefore, conde-

scend to forget their purity, if they would comprehend the pathology of this disease; for the relations of syphilis are so vast and complicated that the physician, as well as the surgeon, must combine their knowledge and their skill, before the many interesting points in the pathology of syphilis can be fully cleared up. To heal the original sore and obtain a cicatrix is but the beginning of the end.

It is partly to this unscientific division of the wide field of medical practice that the phases of opinion regarding the pathology of syphilis have been so remarkably diversified. *The surgeon* alone saw the primary sore or inoculation, and only by chance he might see the development of the future lesions, now so important in pathology. *The physician*, on the other hand, rarely saw the primary sore; and when he now sees the victim of secondary and tertiary syphilis, the case is often extremely complicated, "mixed up with and overlaid by other constitutional and local diseases," which in their turn are made more serious by the existence of syphilis.

After a period, indeed, of skepticism and doubt, we are now confirming, by actual observations (aided by all the advanced knowledge and appliances of the day) the crude surmises of the early physicians regarding the pathology of syphilis.

It was taught by Sir Astley Cooper that "some parts of the body are incapable of being acted upon by the venereal poison, as the brain, the heart, and the abdominal viscera. Indeed," he writes, "this poison does not appear to be capable of exercising its destructive influence on the vital organs, or on those parts most essential to the welfare and continuance of life" (*Lectures on Surgery*). The very reverse of this is now proved to be the truth; and the physicians of the sixteenth century were more advanced in their views than one who was among the most eminent surgeons of the present century.

Toward the close of the fifteenth century a great epidemic of syphilis pervaded Europe, [in the winter of 1494-95, when Charles VIII was besieging Naples], and the historians of the disease described a form of neuralgia as one of the remote results of the venereal poison. In the sixteenth century syphilis was clearly recognized as the result of a specific poison or virus. It was believed to be capable of combining with all other diseases, and so to modify them, and to give them new forms. Even at that early period in medical history, syphilis was recognized as producing phthisis, diarrhoea, dropsy, skin diseases profoundly affecting the system, and demonstrating the presence of a poison in the system by remote general symptoms of ill-health (PARACELSUS). Towards the close of the seventeenth century the ulterior results of venereal disease were fully recognized; but they were believed to be due to bad treatment. Van Swieten taught that *no organ* escapes the influence of the venereal poison. He recognizes it as the source of gummy tumors, exostosis, deep-seated pains, apoplexy, epilepsy, blindness, deafness, paralysis. Benjamin Bell is the first writer on syphilis who puts forth clinical facts in support of his be-

lief that "the venereal disease induces blindness, amaurosis, deafness, phthisis, rheumatism, epilepsy, mania."

There is no disease which more imperatively demands the careful study of the profession at this time, and especially of the army medical officer. The specific distinctions between the "infecting" and the "non-infecting" poison, and the characteristic phenomena they induce, are now being recognized at most of the continental schools. They are distinctions which are of great value in practice, and likely to become more valuable as our knowledge becomes more defined. Even now, indeed, when we see a *primary sore*, AND WATCH IT, we are able to predict with absolute certainty, at an early period of its development, whether the patient will or will not be the subject of secondary symptoms.

A history of syphilis in soldiers is too often the starting-point of a fatal disease. The impairment of the health takes its origin from the date of the *infecting* syphilitic sore. Early implication of the lymphatic glands leads to impoverishment of the blood as an immediate result, and then to the degeneration or wasting of tissues, which attends the general cachexia, and which eventually terminates in death, with complicated and varied lesions especially implicating the internal viscera.

No statistical Nosology gives any idea of the number of men lost to the public service from *syphilis*. The loss of strength from venereal diseases alone is equal to the loss of more than eight days annually of every soldier in the service. Dr. Balfour relates, in his most excellent and interesting *Medical, Sanitary, and Statistical Report of the Army Medical Department* for 1860, that "more than one-third of all the admissions into hospital have been on account of venereal diseases (369 per 1000); and the average number constantly in hospital is equal to 23.69 (reduced to 19.10 in 1864) per 1000 of strength (2315 men), each remaining in hospital on an average 23½ days. Thus the inefficiency is constantly equal to about 2½ regiments." Dr. Balfour also observed the individual history of 1126 men of the Grenadier Guards for three years and five months: 536 of these men gave rise to 1250 admissions; 212 were admitted *once*; 146 *twice*; 70 *three times*; 55 *four times*; 24 *five times*; 19 *six times*; 6 *seven times*; 2 *eight times*; 1 *ten times*; and 1 *fourteen times*.

[In 1861 venereal diseases caused a loss to the state, says Dr. Parkes,* of a period equal to 8.69 days for every man serving at home; of nearly 89,000 men, there was a daily inefficiency from venereal of 2077 men. In 1862, the troops being 78,173 in number, there was a daily inefficiency with venereal of 1739 men. In 1862, of 8.12 days, or equal to the loss of two regiments constantly. In 1863, of 7.4 days. Dr. Balfour calculates that 60 per cent. of the reported venereal cases is syphilitic (recent or remote). In 1862 there were 7771 cases returned in the British army as "*syphilis primaria*," out of a total of 25,789 admissions from "*enthetic diseases*," or at the rate of 30.13 per cent. of the admissions from "*en-*

* [A Manual of Practical Hygiene, by Edmund A. Parkes, M.D., 2d ed., 1866, p. 518.]

thetic diseases." The admissions from primary syphilis, if the diagnosis was correct, would be 99.4 per 1000 of strength; in 1863, 93.8 per 1000 of strength.

The French army statistics are very imperfect, and only the severer cases of syphilis are sent to hospital, so that the total amount of venereal is not readily determined. In 1862, out of a mean strength of 304,733 men in the French army, there were 10,985 admissions into hospital from "syphilis primitive," or at the rate of 36 per 1000 of strength; also, 2636 cases of "syphilis constitutionnelle." Dr. Balfour has calculated that the average non-effective from syphilis in hospital, infirmaries, and quarters, in the French army in 1862, was 11.11 per 1000 serving; while in the British army the proportion in the same year was 10.82 per 1000 serving. But the French reporters state, that on every 5.27 days of sickness from all causes, there is one day from venereal treated in hospitals, infirmaries, and barracks, which would give, if the loss of the services were distributed over the whole army, a loss of 3.9 days yearly for each man. In the British army the loss is between 8 and 9 days for each man—nearly double.

The published statistics of the United States army from 1840 to 1859, give a mean annual rate of only 99 cases of venereal disease per 1000 of mean strength. In the first year of the war, 1861-62, there were 9011 cases, and 12 deaths from syphilis reported; 13,781 cases, and 27 deaths the second, 1862-63. The rates of venereal to the total amount of disease was 1 case of venereal to every 35 taken sick during the first year; 1 to every 41 taken sick during the second.

PREVALENCE OF SYPHILIS IN THE SEVERAL REGIONS.

YEAR ENDING JUNE 30, 1862.						YEAR ENDING JUNE 30, 1863.					
ATLANTIC REGION.		CENTRAL REGION.		PACIFIC REGION.		ATLANTIC REGION.		CENTRAL REGION.		PACIFIC REGION.	
Cases.	Ratio per 1000 of mean strength.	Cases.	Ratio per 1000 of mean strength.	Cases.	Ratio per 1000 of mean strength.	Cases.	Ratio per 1000 of mean strength.	Cases.	Ratio per 1000 of mean strength.	Cases.	Ratio per 1000 of mean strength.
6139	34.51	1628	16.86	1244	185.81	9011	32.05	6118	21.56	6439	21.01
										1224	140.48
										13,781	22.05

Circular No. 6, S. G. O., War Dep't, 1866.]

These figures have great practical significance. They do not tell us how many of these men became constitutionally contaminated by syphilis. They do not tell us in how many the development of "pulmonary lesions of tuberculous inflammation" could be traced to the influence of an "infecting" sore; nor how many afterwards became affected with those lesions of internal organs about to be described, whose characters are now so well defined and characteristic of syphilitic infection.

[Many soldiers die at Netley from various diseases, whose real affection

has been syphilis, as shown by the frequent occurrence of the marks of continued and dominant syphilitic action in their bodies; so that the influence of this cause is very imperfectly indicated by the number of admissions and service lost under the head of enthetic diseases (PARKES, MACLEAN, AITKIN).]

The Director-General of the Army Medical Department very properly requests that "all venereal ulcers be fully described, and the case fully kept;" and if such a request were complied with in the fullest sense of the term, most valuable results would accrue to science. From the nature of the facts and data about to be considered, the great importance of this request will at once appear; and the necessity of describing most fully, distinctly and clearly the origin, development and results of venereal sores, as far as possible, will appear obvious.

With a view to this accurate investigation and recording of results, the following points are worthy of notice:

1. The nature of the contagious principle of the syphilitic poison, as expressed in the opinions of the most trustworthy observers in this and other countries.

2. The characters and the phenomena which distinguish a sore that will contaminate or infect the system, and one which will not.

3. The vehicles or media by which the specific or "*infecting*" virus may be inoculated.

4. The secondary lesions and local growths in the internal viscera which are now so uniformly found to be associated with a history of syphilis, and which are the remote effects of a specific venereal poison.

1. **Nature of the Syphilitic Poison.**—The disease develops itself after the introduction of a specific virus; and the source of the poison is more distinctly traceable than that of the miasmatic order of diseases. The actual substance or matter which contains the virus can be obtained, and can be inoculated. Yet the *active principle* of the poison has not been isolated by any chemical process; and in this respect it is in exactly the same position as the poison of small-pox. The poison of syphilis undergoes a multiple process of elaboration or development in the system before its full effects are completed; and the lesions it induces demonstrate some of the most interesting points in the pathology of the multiplication or reproduction of morbid poisons. It is this multiplication which ultimately destroys life, through a general degeneration of the tissues and the establishment of a cachexia already referred to; or by the induction of grave lesions in important visceral parts, such as the brain, the lungs, the liver, or the kidney.

The earliest effects of the syphilitic poison upon the system become established during the occurrence of a "*hardening process*" which ultimately surrounds an infecting venereal sore—the local papule and its subsequent ulcer or sore. This hardening process is peculiar; and although not constant as to the local sore, it is constant as regards the glands or lymphatics which proceed from the vicinity of the part inoculated. It occurs in one or other of the

three following conditions: (1.) Hardening or induration of *sore* and *glands*; (2.) Hardening and induration of the *cicatrix* and *glands*; (3.) Hardening and induration of *lymphatic glands* only, the original local lesion never having become hard (SIGMUND).

From these specific and characteristic local conditions, as from a focus, the system eventually becomes contaminated. The steps or sequence of phenomena associated with this contamination are not yet clearly understood; but as the contamination is expressed by very constant and specific characters, it is obvious that the original virus has become *intensified* in its action (as is also the case with the virus of hydrophobia), its pernicious influence more active and obvious, while its specific secondary and tertiary effects become more fully and extensively developed. These secondary lesions of syphilis are even now known in some forms to be inoculable.

The following classification and definitions of the forms of syphilis may be given:

A. *Primary Syphilis*. Definition—*Syphilis while limited to the part inoculated, and the lymphatic glands connected with it. The varieties are hard chancre, indurated bubo; soft chancre, suppurating bubo; phagedenic sore; sloughing sore.*

B. *Secondary Syphilis*. Definition—*Syphilis when it affects parts not directly inoculated.*

Tertiary syphilis is a term sometimes applied to the later symptoms, when separated by an interval of health from the ordinary secondary syphilis.

C. *Hereditary Syphilis*. Definition—*Constitutional Syphilis of the child, derived during fetal life from one of the parents.*

Nomenclature.—*Syphilis*, comprehending primary and successive constitutional symptoms of contamination, ought now to be distinguished from simple *venereal* ulceration not followed by gland complication nor contamination of the system. The etymology of the term "*syphilis*" is unknown; but as now used, the term ought to comprise (1) the primary and (2) the successive constitutional symptoms or phenomena which denote the contamination of the system. A man may have had a chancre and a suppurating bubo, and yet remain free from any taint. Such a case should not be set down as a case of syphilis, but simply as a case of "*venereal ulceration*" with *glandular complication*. In such a case the gland complication is consequent on the irritation of the sore, and is not specific.

Syphilis should be reserved to designate the more serious affection, in which the constitution is implicated, and in which the infecting phenomena occur; and if the term "*chancre*" is used, it should be stated* whether it is a "*soft*," "*non-infecting chancre*," or an "*infecting*" one; otherwise, diagnosis is incomplete.

* The following very excellent directions have been drawn up by my colleague, Mr. Longmore, the Professor of Military Surgery in the Army Medical School, to be attended to in recording cases of primary venereal lesions (exclusive of gonorrhœa), and their consequences, among patients in the wards of the Royal Victoria Hospital at Netley. These directions are in accordance with our present knowledge of the Pathology of Syphilis.

"1st. The term '*syphilis*,' or '*syphilitic*,' when used in the case-book, is to be

2. Characters of Venereal Sores, and especially of the "Infecting Sore."

—There are several independent affections to which the common

applied only to such cases as are believed to be of a *specific infecting* kind. Non-syphilitic venereal lesions are to be named according to their local and physical characters, as '*superficial abrasion*,' '*ulcus*,' and the like.

"2d. The following five points are to be noted in entering the history of each venereal case in the case-book :

"I. Physical characters and exact site of the lesions. II. Period of incubation. III. Character of attendant inflammation. IV. Effects on neighboring glands. V. Prognosis.

"3d. Under I. 'Physical characters and exact site of lesion,' state whether,

"(A.) The lesion has the appearance of a papule: fissure: an abrasion: of a dry, or moist, open sore; whether, if a *sore* exists, it is superficial, not appearing to penetrate the whole thickness of the integument: or deeper, with a smooth surface, scanty, chiefly serous secretion, grayish in the centre; whether the texture around the lesions is indurated, and, if so, what is the character of this induration, especially whether it is circumscribed, cartilaginous-like, and appears to be distinct from the subjacent and surrounding tissues; or whether,

"(B.) An excavated sore exists, with abrupt defined edges, involving the whole thickness of the integument, with an uneven surface, covered all over with copious secretion, and without circumscribed induration.

"*Mem.* : The induration which exists from simple inflammation excited by the rubbing of clothes, the probability of which the site of the sore will perhaps indicate, or by the use of irritating applications, such as nitrate of silver, &c., and which disappears gradually in the surrounding tissues, must be carefully distinguished from the circumscribed hardness characteristic of the true syphilitic sore.

"If more than one sore exists, it must be noted whether the several sores appeared together from the first, or appeared in succession.

"If some time has elapsed since the patient was first taken under treatment, the *original* form and appearance of the sore should be traced as far as possible, and noted whether it began as a pimple, abrasion, fissure, or otherwise.

"4th Under II. 'Period of Incubation,' should be ascertained and stated whether,

"(A.) The lesion first appeared after a lapse of one week, or from that time to a month, after exposure to contagion; or whether,

"(B.) There was no period of incubation, the sore appearing within a week after exposure.

"*Mem.* : The importance to the patient of the questions at issue should be frankly explained to him, and his confidence secured, so that he may be induced to state as exactly as he can the number of times he has been exposed to contagion within a period of four or five weeks prior to his discovering the existence of the lesion. A patient usually himself dates the origin of the lesion from the time when he was *last* in the way of contracting disease. He may, however, have been in the way of contracting disease many times *after* the particular occasion on which he really contracted it.

"5th. Under III. 'Character of attendant inflammation,' state whether,

"(A.) The inflammation appears to be of the adhesive, or whether,

"(B.) Of the suppurative or phagedenic kind.

"6th. Under IV. 'Effects on neighboring glands,' state whether,

"(A.) The superficial inguinal glands are, on one or both sides, generally and separately indurated, the inflammation with which they are affected being of an indolent character and without pain; or whether,

"(B.) The glands are free from enlargement; or whether one or more of the glands are enlarged, and exhibit a tendency to suppurative action.

"7th. Under V. 'Prognosis,' state whether you consider the case to be one of (a.) syphilis, or (b.) of local venereal sore, or (c.) of a doubtful nature.

"If the circumstances described under (A.) exist, the conclusion will be that the lesions are indicative of the constitution being affected by syphilis; if those described under (B.) exist, the conclusions will be that the lesions are local.

"If your prognosis is doubtful, state the considerations which cause it to be so.

"If the prognosis that the patient is afflicted with syphilis be correct, then the specific sore will not be capable of repetition on the same person by inoculation; if

name "Venereal" has been applied, each capable of transmission from person to person within certain definite periods. From time to time it has been a subject of discussion, "Whether these *several affections* are due to one and the same virus, whose action is modified by admixture with secretions, or by peculiarities of constitution on the part of the recipient?" or, "Whether a separate specific poison exists for each form of venereal disease?" This latter alternative is now proven to be true; and the following are the classes of venereal affections which are specifically distinct: (a.) Gonorrhœa: (b.) "Simple" "non-infecting" chancres, ulcers, or sores; (c.) "Infecting" chancres, papules, ulcers, or sores; (d.) Mixed chancres—the combined result of the virus of (b.) and (c.); (e.) Subsequent lesions retaining specific powers of contagion (some forms of secondary syphilitic lesions).

The history of the identification of the nature of the separate poisons which give rise to the several venereal affections arranges itself into three periods as to time, and is comprehended in the medical records of the past century.

I. *The Period and Doctrine of Hunter—The Hunterian Chancre.*—Hunter taught the doctrine (now known to be an error), "That the various forms of syphilis and gonorrhœa depend upon one and the same poison—that the matter or virus produced in both is of the same kind, and has the same properties."* He believed that he had established, by experiment and observation, that the discharge from a gonorrhœa will produce either a gonorrhœa, or a chancre, or the constitutional affections of syphilis—and that the matter from a chancre will indifferently give rise to either of these venereal affections. Hunter rested his belief and his doctrine mainly on an experiment on himself. He dipped a lancet in the venereal matter from a gonorrhœa. He made two punctures in the tissue of his own penis with the lancet so charged. One inoculation he made on the *glans*—the other on the *prepuce*. Two distinct results followed, *each of them marked by a distinct and specific period of incubation*. The inoculation on the *PREPUCE* was followed by itching from the *third to the fifth day*. On the fifth day the site of the puncture was red, thickened, and swollen. A speck became visible; and in a week this speck had commenced to suppurate; the urethra at the same time indicating the commencement of a

the prognosis be correct that the sore is a simple one, then the sore will be capable of indefinite repetition on the subject of it by inoculation.

"If your prognosis is doubtful, regard the disease as local until further observation establishes a contrary opinion."

* [Up to the beginning of the eighteenth century the oneness of the virus of gonorrhœa and syphilis was generally held; or, rather, gonorrhœa and syphilis were confounded as of common origin. The first who combated this doctrine, and maintained the non-identity of the two disorders, were Cockburne, of London (*The Symptoms, Nature, Cause, and Cure of a Gonorrhœa*, London, 1715), and Balfour, of Edinburgh (*Dissertatio de Gonorrhœa Virulenta*, Edin., 1767). Benjamin Bell (*On Gonorrhœa Virulenta and Venereal Disease*, London, 1793) challenged Hunter's doctrine, and opposed it by a series of observations and experiments; and was the first to point out the occasional presence of specific ulcers in the urethra, thus showing one of the sources of the error into which Hunter had fallen.—EDITOR.]

discharge. The inoculation on the GLANS was followed by itching *fourteen days after the puncture was made*: three days later a speck appeared where the puncture had been made. The speck became a papule, then a pimple, and ultimately discharged yellow matter. The sore on the prepuce broke out several times after it healed up; but the sore on the glans never broke out again after it healed. The secondary lesions of syphilis followed this experiment, demonstrating the "infecting" nature of a virus with which he had been inoculated. Ulceration of the throat commenced in due time, and copper-colored blotches on the skin followed in the usual sequence. The time the experiment took, from the first infection to the complete cure and elimination of the poison, was three years.

Now, with the knowledge of syphilis which we possess, can we say from which of these sores the constitutional disease arose? The answer will evolve itself in the sequel. Hunter believed he had inoculated the discharge of a specific gonorrhœa *only and alone*; but two important questions now suggest themselves, concerning which Hunter does not enlighten us, namely: Had the person a concealed infecting chancre from whom Hunter took the virus? Was the patient suffering from constitutional syphilis at the time he had a gonorrhœa?

Besides Hunter, Carmichael in this country taught the same doctrine of a *single virus*;* and Cazenave in France.

II. *The Period and Doctrine of Ricord*.—Ricord established, by numerous experiments repeated in various ways,—(1.) That the inoculation of gonorrhœal discharge by the skin is followed by no specific result; (2.) That *at least two*, if not *three*, distinct poisons exist—namely, *one virus* which would produce a gonorrhœa—another *virus* which would give rise to a specific ulceration, called a chancre. The ulceration of a chancre he observed to follow a very definite course. It commenced, as a rule, within twenty-four hours after the inoculation of the poison. A pustule formed, which breaking, a *soft* or suppurating chancre was the result. Ricord, however, eventually recognized *two classes* of chancres—the *soft* and the *hard*; but he described them as originating in the same way,—by contagion from a similar primary sore. His experiments were of one or other of two kinds. Either they were made on persons who had been already affected by syphilis—now known to involve a most

* [Carmichael maintained the doctrine of a plurality of infecting viruses (*An Essay on the Venereal Diseases which have been confounded with Syphilis*. By Richard Carmichael. Dublin, 1814). He is claimed, though wrongly, by the dualists, as the father of their doctrine; for, besides several continental writers, including Swediaur, Abernethy (*Surgical Observations on Diseases resembling Syphilis*, 1804) speaks of contagious ulcers of the genital organs not followed by constitutional symptoms. The absence of induration of the base, and a tendency to slough, he particularly mentions as their chief characters. In the third edition of the same work (1814) he remarks, "I have never seen the phagedenic ulcer, which suddenly sloughs, affect the constitution." Carmichael divided ulcers on the genital organs into *syphilitic* and *venereal*, and of the latter he makes four subdivisions. (1.) Superficial, edges elevated, non-indurated; (2.) The same, without elevated edges; (3.) Phagedenic; (4.) Gangrenous. He expressly states, too, that these are sometimes followed by constitutional symptoms, and gives examples.—EDITOR.]

vital fallacy in drawing conclusions regarding the nature of syphilis; or on persons concerning whom it was not ascertained whether they had been infected with syphilis before or not.

Hunter showed that the secretion from *one* kind of syphilitic sore is not capable of being inoculated on the same body that produced it; and now we know that the discharge from the "infecting" sore cannot be inoculated on the already infected person. Ricord has further shown that the *plastic lymph*, the increased growth of tissue round a true chancre—the *specific sclerosis* or induration—does not take place a second time on the same subject; while Sigmund and many other observers are now agreed that the "infecting" disease does not repeat itself. This brings us to—

III. *The Present Period in the History of Syphilis.*—Its commencement is of very recent date—since 1856; and is characterized by a belief in the *duality* of the venereal virus exclusive of gonorrhœa.*

The surgeons of Lyons—Rollet, Diday, and Viennois—Mr. Henry Lee, of the Lock Hospital, and Mr. Henry Thompson, of University College Hospital in London, Hubbenet, of the Syphilitic Clinique at Leipsic, Sigmund, of Vienna, and Von Bärensprung, of Berlin, are those who, by experiment and careful observation, have thrown most light on this remarkable disease. In addition to the specific virus of gonorrhœa (which may now be eliminated as distinct from those about to be noticed), these observers recognize two forms of venereal disease, distinct in their origin, propagation, and development. They recognize specific differences in the mode of development, and in the sequence of phenomena which distinguish an "infecting" and a "non-infecting" sore. They have shown that the sore which eventually *contaminates* the system commences differently from the sore which does not infect the system. The "infecting" sore (the one which contaminates) commences as a papule, pimple, abrasion, fissure, or crack, around which a specific growth of tissue takes place—a *sclerosis* or *induration*. A pustule is no essential part of the process, nor is suppuration. They are accidental phenomena, the result of irritation, pressure, or laceration, which produces a sore or ulceration—a result always very easily established and maintained in connection with infecting sores, as compared with other sores.

* [This is not the place to discuss the doctrines of the unity or plurality of the syphilitic virus. The Commission appointed by the Lords of the Admiralty to inquire into the venereal disease, with the view to diminish its effects in the British Army and Navy, in their report, which is founded on the evidence of all the eminent members of the profession in Great Britain, on the question of the unity or duality of the virus, hold this language: "There is probably but *one* true syphilitic poison exerting its influence upon the soil in which it is implanted, producing various forms of true syphilitic sores, differing in different individuals, modified by health, and by constitution, by locality, and probably by its ever-varying intensity." Mr. Hutchinson (Reynold's *System of Medicine*, vol. i), well remarks: "The doctrine of the so-called duality of syphilis seems to rest on the most unsubstantial foundation. Surely it is absurd to speak of the duality of things which have scarcely any features in common." There is no more reason to believe in the existence of two poisons in syphilis than in small-pox; and the absurdity of the doctrine, so inconsiderately accepted by a portion of the profession, is beginning to be generally appreciated.—EDITOR.]

In women, compared with men, the open sore is said to be still more rare as the form of "infecting" sore. A hard chancre or sore in them is exceptional; and when it does occur, it remains small, is ill-developed, and is readily overlooked, even when searched for with great care, aided by a vaginal examination with the speculum.

[The induration disappears too at an earlier stage in women than in men, hence the *age* of the sore at the time of examination should be noted. The rapidity with which well-marked induration often passes away in the female, has been noticed by Clerc; he has also remarked that in certain regions it is more developed than in the same parts in men—as the lips and orifice of the urethra.]

In them the primary lesion which *infects* the system is always a papule (SIGMUND, CLERC). Another peculiarity connected with the "infecting" sore in women is, that such papules are apt to form along the course of the superficial lymphatics; and Ricord admits that induration is generally absent or ill-developed in primary sores in the vagina.

When the papule opens and becomes a sore, the fluid discharged from its open surface has been shown by Hubbenet, Lee, and Rollet to furnish a diagnostic test of the kind of disease, and of the sore from which it proceeds. Sigmund does not go so far as this. He does not consider the sores or chancres so different in form or character as to be at once distinguishable the one from the other. *He waits to see the virus produce PART of its effect* upon the system beyond the site of inoculation before he decides as to the nature of the sore. He waits to see the lymphatics indurate. He believes that then, and not till then, the distinction can be absolutely drawn between a sore which will infect the system and one which will not. He believes—(1.) That if induration of the lymphatics does *not* take place within six or eight weeks, and (2.) That if repeated successful auto-inoculation can be made on the bearer of the chancre during this period, then it is certain that the sore will not infect the system. If, on the contrary, the lymphatics indurate, and auto-inoculations cannot be then effected, the sore is assuredly an "infecting" chancre.

The addition to our means of diagnosis from the nature of the discharge—pus from the one, not from the other—is one of great value when it can be made, because the diagnosis as to the probability of subsequent infection may in some cases be made earlier.

Period of Incubation.—The time of the commencement of a sore or lesion after inoculation or contagion is of great importance to be noticed. A definite period of incubation exists for the "infecting" sore, fixed by experiment as well as by casual observation. Diday and Rollet fix the period at twenty-four days if the poison is from a primary sore; but at twenty-six days if the poison is from a secondary lesion. Sigmund, of Vienna, fixes the period of incubation at from fourteen to twenty-one days. Sometimes it may be longer, but never beyond six weeks or forty-two days. The circumstances which may protract the period thus long are exhausting fevers, pregnancy, and anæmic states of the constitution.

[The mean period of incubation in twenty cases observed by Diday, was fourteen days;* in forty-six cases analyzed by Fournier, thirty-two;† in fourteen cases observed by the latter, the incubation was respectively, seventeen days, twenty-one days, over three weeks, twenty-five days, twenty-eight days, four weeks, three weeks to a month, thirty to thirty-five days, thirty-three, thirty-five, and thirty-six days, thirty-three to forty days, and, finally, seventy-two days; in the last case he was able to satisfy himself of the accuracy of the facts.‡ Rollet analyzed and grouped twenty-eight cases after inoculation, in which the time of incubation was precisely ascertained; the mean was twenty-five days.§]

Contamination of the System.—The “infecting” sore does not remain merely a local disease. It contaminates the system, giving rise by zymosis or multiplication to one of the most malignant, and most lasting, and most destructive forms of a poison-disease that affects the human frame. How is this brought about? The only constant index of such contamination or secondary disease commencing, seems to be the occurrence of multiple enlargement of related lymphatics and lymphatic glands, which begins about ten or twelve days after the papule, ulcer, or chancre has made its appearance; or from four, five, or six weeks after contagion, or inoculation by sexual intercourse, or otherwise. This, too, may be delayed by exhausting diseases till three months after contagion. Such glands do not suppurate. They enlarge slowly, and without pain in the immediate vicinity of the sore; and eventually those [the epitrochlea and] in the axilla become similarly affected, and ultimately enlargement of the chain of glands extending up towards the occiput, behind the sterno-mastoid muscle, is apparent. A general morbid condition of the whole system is the necessary result of this extensive disease of the lymphatic glands. Nutrition becomes defective. The blood is changed; it becomes anæmic. Emaciation is then often rapid. The digestive organs are impaired in function. The muscles lose their hardness, elasticity, and energy; and the lesions peculiar to syphilis set in.

[Shakspeare has well described the effects of syphilis in Timon's vituperation, addressed to Phrynia and Timandra.

. season the slaves
For tubs and baths; bring down rose-cheeked youth
To the tub-fast and the diet.

. Consumption sow
In hollow bones of man; strike their sharp shins,
And mar men's spurring. Crack the lawyer's voice,
That he may never more false title plead,
Nor sound his quillots shrilly; hoar the flamen,
That scolds against the quality of flesh,
And not believes himself; down with the nose,
Down with it flat; take the bridge quite away,
. make curl'd-pate ruffians bald;

* [Gaz. Méd. de Lyon, 1858.

† Recherches sur l'incubation de la Syphilis, 1865.

‡ Gaz. des Hôpitaux, 1865.

§ Traité des Maladies Vénériennes.]

And let the unscarred braggarts of the war
Derive some pain from you.

TIMON OF ATHENS, *Act iv, Scene iii.*]

The following table exhibits a scheme of the periods of appearance of the phenomena after inoculation from an infecting sore, and estimated from the first appearance of the papule or sore (SIGMUND):

		Day—9th 10th 14th 17th 19th 21st											
I. INDURATION OF SORE, .	Week—4th	5th	6th	7th	8th	9th	10th	11th	12th				
	Cases—31	44	56	74	46	20	13	9	—				
II. ENLARGEMENT OF GLANDS, .	"	—	2	41	68	45	22	11	—	2			
III. SPOTS ON THE SKIN, .	"	—	—	—	3	10	11	24	27	31			
IV. PAPULÆ AND PUSTULES, .	"	—	—	—	7	22	34	41	41	42			
V. AFFECTIONS OF FAUCES, .	"	—	—	—	—	—	—	—	—	—			

Cutaneous Affections.—Besides the general involvement of the glands, the condition of the skin may further demonstrate the contamination of the system. In the more insidious form of contamination its color generally is altered. It becomes pale, white, fawn, yellow, or brown; and is wrinkled, dry, harsh, rough, and hard, and no longer soft and elastic. The eruptions are papular, pustular, or scaly, and they are peculiar in their symmetry of distribution, and in the curvilinear character of their grouping. They leave behind them stains of color, pale cicatrices, or persistent ulcerations of the true skin. The local distribution of the syphilitic eruptions are also peculiar (DEVERGIE). "Their seats of election in the order of frequency are,—(1.) The parts round the alæ of the nose and the angles of the mouth; (2.) The roots of the hair at the forehead and back of the neck; (3.) The inner angle of the eyes; (4.) The centre of the breast; (5.) The inner side of the limbs, the neighborhood of the axilla and the groins."

The Affections of the Fauces are often not more than a peculiar color of the mucous membrane, persistent, however, like the staining of the skin (GAIRDNER), and eventually leading to disorganization. In women the process may cease with a slight follicular swelling of the mucous membrane of the fauces, tonsils, and soft palate (SIGMUND). If the process does not cease, then superficial erosions or deep ulcers of the soft palate supervene. Or still more diffused forms of ulceration may set in, involving great destruction of parts, and spreading in all directions—encroaching on the nasal fossæ and pharynx, eating away the epiglottis, extending down the air-passages, and even causing necrosis and exfoliation of the cartilages of the larynx.

Second Attacks of Syphilis.—The general infection is of such a kind as to render the system, as a rule, proof against a second invasion of the specific "*infecting*" virus. The disease never repeats itself, except, it may be, after a long interval. Sigmund has seen such a case. In this respect it resembles other virulent diseases acknowledging a specific virus as their origin; and in them the immunity is usually, but not invariably, complete—*e. g.*, small-pox, cow-pox, scarlet fever, and the like.

[This statement should not, in the present state of our knowledge, be

unreservedly admitted. A certain immunity is no doubt given by one attack of syphilis; but it is relative and not absolute, and depends upon contingencies. Of thirty-three witnesses examined by the Venereal Commission, twenty-three asserted that one attack of syphilis gives no future immunity. Mr. Hutchinson (Reynold's *System of Medicine*, vol. i) has lately seen two attacks in the same person, and says, "the disease was slight the second time, and appeared to have undergone such modification as is usually observed in small-pox after a successful and recent vaccination." The infrequency of the typical hard chancre in prostitutes is well known, and this is due, probably, in a measure, to a certain protection given by a previous attack. The question of inherited immunity, or the influence of the disease in the parent, giving protection, partial or complete, to the offspring, is one of great interest, and has been investigated and treated of by Mr. Hutchinson (*London Hospital Reports*, vol. ii, 1865; Reynold's *System of Medicine*, vol. i, 1866). As the disease itself is transmissible, so may be the immunity, for a certain time, at least, against a second contagion. Clinical experience would seem to favor the view, that those who have suffered severely from the inherited disease are to some extent protected. Mr. Hutchinson remarks that, "in the history of congenital syphilis nothing is more common than to meet with instances in which the eldest child of a family suffered severely in infancy, the second less, and the third still more slightly, and the others not at all." If the eldest enjoys immunity, the degree of protection will diminish in ratio to the distance from the original taint; slighter degrees of immunity being shown in a milder form of the disease, as in small-pox after vaccination. Mr. Hutchinson asks if it is not "probable that a very considerable portion of the community being the descendants of those who have suffered, enjoy in a certain degree, infinitely slight in many, but powerful in others, immunity from further attacks." Mr. Henry Lee (*Lectures on Syphilis*, p. 209), is of opinion that "a person who has had hereditary syphilis in his youth will either not contract the infecting form of syphilis in after life, or will have it in a modified form." According to the late Dr. Ferguson, the Portuguese explained the mildness of syphilis amongst them by the acquisition of hereditary immunity. Still cases are met with, where persons who have had hereditary syphilis have, at a later period of life, contracted true chancres. Mr. Hutchinson himself mentions the case of a subject of inherited taint, who contracted a chancre, and which was followed by constitutional symptoms, and presented an example of acquired and inherited syphilis, present at the same time in the same person.]

After the system is once infected, the specific sore cannot be transplanted by contagion or inoculation to any other part of the body. The "infecting" sore is not *auto-inoculable*; and a person suffering from a chancre infecting his system will not be affected by a further inoculation of the same specific virus. There is, however, a slight qualification to be made here. Mr. Henry Lee has shown that (1.) There is a *stage* in the existence of an "infecting" sore when it is *auto-inoculable*. That period or stage is a very early one in the existence of the sore—namely, before any specific systemic action has begun to develop. If at this period the poison of another "infecting" chancre from another person, or from the chancre already existing on the same person, be inoculated, then a second "infecting" chancre, accurately representing the original, will result. The period when this event can happen is *before* the gland induration.

(2.) Mr. Lee has shown that there is a *certain condition* of the chancre in which, at any stage, on being inoculated or transplanted, it will produce a sore. It then appears to be autoinoculable. But this is only in appearance, and not in reality. The condition of the chancre that does this is one of *irritation*. Blister a chancre, or irritate it by an irritating ointment, or by any other means, so as to cause pus to flow—free pus-corpuscles being generated—and then we may have what has been recently termed a “mixed chancre,” of much more frequent occurrence than has generally been supposed (SIGMUND). Sigmund has produced such chancres by inoculation. The utmost caution, therefore, is necessary before pronouncing a sore to be *non-syphilitic*—i. e., “*non-infecting*.” Sigmund inoculated the pus of a soft, contagious, or suppurating sore upon the infiltration or sclerosis of a hard papule on which the skin had remained unbroken. Between twenty-four and forty-eight hours after, a suppurating ulcer was established, which afterwards assumed Hunterian characters. Inoculation of two poisons may thus be in some cases simultaneous or successive. Hence, “mixed chancres” present two aspects: on the surface is the soft, contagious, pus-producing ulcer; while deeper down is the specific syphilitic infiltration of the true “*infecting*” virus. Local plugging and enlargement of the superficial absorbents take place from such “mixed chancres,” followed by similar infiltration of the group of lymphatic glands nearest to the sore, spreading gradually to distant and more distant groups. This is the constant series of phenomena after syphilitic “infection,”—a regular series of connected events, giving rise to such symptoms as are associated with no other disease-poison except that due to syphilis.

In many cases of sloughing phagedena the syphilitic poison at once induces slough, as is the case with the poison of snake-bites, already referred to at page 647, when the tissue and the virus both die simultaneously. Such cases do not, as a rule, infect. So, also, if the part inoculated is made to slough by escharotics, both the virus and the tissue may be destroyed; and herein lies the value of the early use of caustic sufficiently powerful to destroy the inoculated part.

On the other hand, the sore which does not infect, and which does not contaminate, is the “soft,” suppurating sore—the “chancreoid ulcer,” as it has been called—or “the simple contagious ulcer of the genitals.” The virus begins to act from the very moment of its application, and, after the formation of a pustule, ulceration is generally established by the sixth or eighth day from the time of infection, [or sooner.]

[The sore is characterized by active ulceration and suppuration, its edges and base are discontinuous, the vertical edge as it joins the base being a little undermined, and the former can be slightly moved on the latter, a little pus oozing out. The base is cellular and honeycombed, and the sore, when felt between the finger and thumb, is soft and doughy. There is never marked or well-defined specific induration. Rarely more than one or two inguinal glands are affected.]

It is purely a local disease, and is generally very soon accompanied by an enlargement of the lymphatic glands, which goes on to suppuration, and ends there. This sore may be transferred or transplanted at will, by contagion or inoculation, from one part of the body of the patient to another, or from one person to another. It is thus auto-inoculable, and is always so, the period of incubation being short—about twenty-four hours only. Its virus is particularly irritating. Hence numerous sores of this nature may exist on a person at one and the same time; but *successive infecting sores* do not, as a rule, ever exist on the same person. The *multiple* character of the simple sore is now generally recognized, and likewise the *solitary* character of the infecting one.

[Of 456 hard chancres observed by M. Ricord in 1856, 341 were single, and 115 were multiple (*Leçons sur le Chancre*, 1857), or three-fourths. Clerc found in 267 men suffering from constitutional syphilis, the chancre single in 224, and multiple in 43, or four-fifths.]

The soft chancre is altogether a local sore, and so remains; while thousands of them may be multiplied at will successively over the same person's body, especially during its stage of suppuration and ulceration. The ordinary site of the soft chancre is on the *prepuce*, and in the sulcus behind the *corona glandis*. The parts most susceptible of laceration are the parts most exposed to the inoculation, and where the virus is most likely to nestle and to be overlooked (MILLER).

As far back as 1856, Mr. Lee showed, and Mr. Rollet since then has also shown, that not only is the infecting sore not capable of being transmitted from one part of the body to another, but it is not inoculable upon a person who has been already contaminated by syphilis, more particularly so long as lesions continue to develop themselves. It is, therefore, as necessary now to distinguish "infecting" and "non-infecting" sores as it is necessary to distinguish the various forms of continued fevers.

In future experiments and observations as to the effects of primary syphilis, it must be remembered that the subject cannot be studied or experimented with to any extent, upon the patient himself. For this reason many of the early observations of Hunter and Ricord are limited and fallacious: one might as well attempt to study the vaccine disease by re-inoculation of it on the same person a second time, immediately after it has produced its specific effects.*

* Another kind of sore, very frequently seen about the *prepuce* or *glans penis* of men, or the *vulva* of women, is mentioned here to guard against its being confounded with soft venereal sores or with chancres—I mean the sore which forms after the eruption of *Herpes preputialis*. The eruption of *Herpes preputialis* commences by itching, which is felt at the base of the *glans*, at the internal surface of the *prepuce*, or at the junction of the *prepuce* and the *glans*. This itching is often so slight as not to attract notice, and it is not of the same kind, nor does it occupy the same site, as the itching of gonorrhœa. The surface of the *glans* or of the *prepuce* may be red, and although nothing may be at first visible with the naked eye, yet by

[The received views on these points may be thus summarized:—(1.) A true chancre is an excoriation or ulceration produced by a specific virus at the point of deposition or inoculation, and is the antecedent of constitutional syphilis in the person so affected. (2.) There is a constant, though variable lapse of time between the inoculation and the appearance of the local sore—period of incubation—whose mean duration varies from twenty-two to thirty-five days. (3.) In a large majority of cases a true chancre begins with an erythematous inflammation at the contaminated point, which is immediately followed by a diphtheritic product *sui generis*, especially when its site is a mucous membrane. At times it is a flattened induration, or an enlarged papule, covered, perhaps, with an adherent scale (H. LEE). Induration of the base of the sore is very common, and is held by many to be pathognomonic; but this is an error, for it may not exist either as an early or subsequent symptom, as in chancre of the integument of the penis, and of the genital organs of the female; or it may be slight from the onset, and soon disappear; or it may not be detected; or specific induration may not be distinguished from hardness, the result of other morbid processes. Induration happens in most cases at the end of the first or beginning of the second week; it may appear later. It is fibro-plastic and presents different characters according to its age and the site of the sore. At the onset it is slight and diffuse; at the period of maturity of the chancre it is usually marked, deep, well-defined, hemispherical like a pea, and extends beyond the limits of the sore. In the ulcerous sore the edges slope inward to the base and are continuous with it. The typical form of true chancre may then be stated as beginning in a pimple or papule, which soon becomes an induration, and for some time is non-ulcerative, but covered with a gummy sero-epithelial secretion, and then taking on molecular disintegration at the surface. The induration is indolent, and is dissipated by a process of absorption, and not by liquefaction of the interstitial tissue-material. The formation of pus is an epiphenomenon from accidental irritation. A cicatrix is rarely left after the erosive form of true chancre. Four times out of five true chancre is single; if multiple it is so from the first. Except under circumstances of attendant irritation, the matter of a true chancre is not auto-inoculable, and then produces only pustulation and erosion without induration. Its duration varies accord-

means of a hand lens small circular elevations of cuticle are to be seen, raised by limpid serum. Several groups of these small vesicles generally occur, separated from each other by the space of a few lines. These eventually burst, and in their place small circular ulcerations, perfectly distinct, are to be seen, with a red bottom, and measuring scarcely a quarter of a line in diameter. The site of these herpetic ulcers is highly sensitive, and secretes pus and fluid, usually of an offensive and peculiar odor.

When the groups of vesicles are situated on the *cutaneous* surface of the prepuce, they are but slightly inflamed, compared with those situated on its internal or mucous surface. Frequently the fluid contained in the vesicles on the cutaneous surface is re-absorbed, when slight desquamation ensues over the lesion. If the fluid is not absorbed, it becomes opaque after a few days; and small scaly incrustations take the place of the group of vesicles. The disease may thus terminate in about *seven or eight days*.

When the groups of vesicles form on the internal aspect of the prepuce, they increase in size rapidly, and the inflammation is much more active. The walls of the vesicles are so extremely thin and transparent that the red color of the inflamed tissue may be seen through them. But the fluid soon becomes opaque and sero-purulent, small moist crusts or scabs form, which, being detached naturally or accidentally, expose excoriated spots, and it is important to distinguish these from soft venereal chancres. The venereal sores never commence as vesicles.

ing to its extent, the degree of induration, and whether erosive or ulcerous; it is usually from fifteen days to three weeks. A large ulcerous chancre, with deep-seated induration, may last two or three months. The influence of treatment on its duration is decided.

Though chancre is met with in every part of the human body that can be seen or handled, its most common site is the genital organs (95 per cent.), and the special sites in them are those parts most liable to excoriation, or where the specific virus can most readily rest, as the cervix penis and mucous surface of the prepuce in the male, and the labia in the female. The site of hard chancre, in 403 males noted by Clerc, and in 361 males noted by Bassereau, was as follows:

	Clerc.	Bassereau.
Internal surface of prepuce and cervix,	234	216
Free border of prepuce,	34	10
Frenum,	14	35
Glans penis,	12	20
Meatus,	33	14
Integument of penis,	58	36
Peno-scrotal angle,	5	0
Scrotum,	3	6
Lips,	5	12
Eyelids,	1	0
Gums,	0	1
Tongue,	1	4
Pubes,	2	2
Thighs,	1	1
Buttock,	0	1
Anus,	0	1
Cheek,	0	1
Thumb,	0	1
	403	361

In 730 hard chancres situated on the genital organs 450 were on the mucous surface of the prepuce—60 per cent.

In 113 cases of hard chancre in the female, where the site was noted by Clerc, he found in

The labia externa,	25	Thighs,	2
The labia interna,	29	Lips,	7
Fourchette,	16	Tongue,	1
Neck of uterus,	1	Base of uvula,	1
Meatus,	2	Mouth,	1
Vestibulum,	2	Nose,	3
Perineum,	4	Forehead,	2
Anus,	7	Neck (left side),	1
Genito-crural fold, and groin, 6			

113*

(4.) From one week to one month after the local development of the virus, the glands, which receive directly the lymphatics of the part primarily affected, become symmetrically enlarged and indurated—as in chancres of the penis and vulva, the superior chain of inguinal glands. Acute or suppurative adenitis is not common. The lymphatics may become enlarged and tender, but angeioleucitis is rare. (5.) When induration of the base of a true chancre exists, it is by many, and probably

* [The absence of any chancre of the vagina in the above record is curious, and must be attributed not only to its being a very infrequent site of the hard sore, but to the great difficulty in thoroughly exploring that region with the specula in common use.—EDITOR.]

rightly, regarded as the first of the constitutional symptoms,* "the prelude of the diathesis and the local reaction of the general poisoning" (WILKS). Not infrequently, after the local sore has lasted two or three weeks, rheumatoid pains, headache, weariness, &c., are complained of. These are early and sure tokens of systemic infection. They are very commonly followed, in the course of from four weeks to two months, by symmetrical exanthems on the skin and mucous membranes, symmetrical affections of the nails, hair, eye, and later, unsymmetrical ulcerations in the mouth, throat, and skin, tending to spread widely and deeply, with fibro-plastic exudations of the periosteum, connective tissue, muscles, fascia, nerves, viscera, not usually symmetrical, chronic in progress, and attended often with ulceration, or even a sloughing disposition, with tendency to relapse; for when the virus has entered the system, there is scarcely a tissue that may not be implicated, and that always in a specific and characteristic manner,—by the exudation of fibro-albuminoid material, modified to some extent by the organ in which it happens; in the solid organs as circumscribed masses, whilst on free surfaces it is seen on the base and borders of ulcerous sores, the same as in the primary local lesion. There is quite often entire freedom from any symptoms, lasting for months and even years, as if the virus had been exterminated; but usually certain reminders, in the form of scattered, scaly patches on the skin, as psoriasis palmaris, sores on the tongue, lips, &c., appear from time to time. So long as this tendency or state exists it is evidence of the presence of a virus in the system, communicable by direct or indirect means. Either from the prolonged effects of the special toxic agent upon the constitution, or from other concomitant causes, a cachectic condition may come on at a later period, varying from a few months to twenty years, with a tendency to fatty degenerations of the various structures of the body, and, perhaps, to those known as waxy or lardaceous. These are the so-called tertiary symptoms, but are more properly the sequelæ of syphilis. (6.) True chancre gives a *relative* and not *absolute* protection against subsequent attacks of the malady.]

3. Vehicles or Media by which the Specific "Infecting" Virus may be Inoculated.—Besides the discharge (non-purulent or mixed) from an infecting sore, there are at least three other sources of infection, namely,—(1.) The contagion of secondary syphilitic sores—*e. g.*, the syphilitic secondary ulceration of the female nipple, inoculating the mouth of the healthy infant born of healthy parents. (2.) It is now also established that *secondary* syphilitic inoculation (*e. g.*, the discharge from the softening and ulceration of gummatous tumors, mucous tubercles, papules, and the like) gives rise to a sore which exactly resembles a primary infecting chancre (LEE, ROLLET, VIENNOIS). But it is said to differ in the following particulars, namely,—(a.) The period of incubation is said to be somewhat longer; (b.) Ulceration is superficial; (c.) The sore heals in a shorter time; (d.) Induration is less marked; (e.) The constitutional infection is longer in developing itself; and (f.) The lesions which result are said to be not again contagious. Thus it is supposed the great epidemic of the fifteenth century gradually abated. Hence, also, perhaps, the modern belief in the modifying influence of syphilization may to some extent be explained—an operation which is not

* [Ambrose Paré said, "If there is an ulcer on the penis and the part is hardened, it will be an infallible sign that the patient is affected with (constitutional) syphilis."]

warranted by the present scientific knowledge we possess. (3.) The blood of those suffering from acute secondary syphilis inoculates. (4.) A female, otherwise free of syphilis, may become contaminated during the gestation of a fetus begotten of a male who, at the time of the fruitful connection, was himself alone suffering from contamination of the system by syphilis, in some form of active secondary phenomena (DIDAY, MACLEAN). In such cases no syphilitic sores existed on the genital organs of either party at the period of sexual intercourse.

[The vehicles by which the syphilitic virus may be communicated to a healthy person, are innumerable. Primary disease of the pharynx has been caused by catheterism of the Eustachian tube (FOURNIER, BUCQUOY, CULLERIER, LABOULBÈNE). Cases of infection have happened amongst glass-blowers, barometer and thermometer makers; as well as by the use of contaminated sponges, syringes, drinking-glasses, linen, washing-tubs, surgical instruments, &c. Evidence is conclusive to the effect that syphilis may be communicated by sexual intercourse during its constitutional stage (*Venereal Commission, ante*). With regard to the inoculability of the disease by vaccination, the question and conditions are thus fairly stated by Auspitz: (1.) Vaccination may give rise to the inoculation of syphilis; (2.) We are not yet in a position to positively state when syphilis exists in combination with the vaccine virus; (3.) This power of transmission exists in the virus itself, though the lymph cannot be looked upon as a syphilitic product; (4.) It is rather ordinary lymph with syphilitic properties engrafted on it during its passage through a syphilitic system; (5.) An intimate molecular change does not happen, there being merely a mechanical mixture of the vaccine and syphilitic products (*Die Lehren vom Syphilitischen Contagium*, p. 270, Vienna, 1866).]

Experiments at Florence, at the Clinique for venereal diseases, show that healthy persons may be inoculated with the blood of syphilitic patients. It is related that, on January 23, 1860, two young doctors were inoculated with the blood of a syphilitic patient, but no result followed; on February 6, 1862, three other doctors (perfectly free from syphilis) were inoculated by venous blood taken from a female suffering from the acute lesions of secondary syphilitic disease. Charpie soaked in the blood was applied to an abrasion in the arm of each. On March 3 (twenty-five days after the operation) a slight itching and elevation was perceptible, a papule formed, which eight days afterwards, became covered with a crust. This crust increased in thickness day by day, and twelve days after the appearance of the papule two glands in the axilla became enlarged, and the sensibility of the papule increased. Nineteen days after its appearance the crust fell off, leaving a funnel-shaped chancre, with elastic resistant borders. On the twenty-third day the chancre had increased in size and induration. On the fortieth day eruption on the skin and glandular swellings in the neck supervened. The erythema lasted eight days, and pursued a regular course.

On the forty-eighth day the glands had increased in size and hardness, the chancre maintaining its specific condition, showing no tendency to heal.

On the fiftieth day the color of the erythema became decidedly coppery, and treatment by mercury was then begun. In these experiments the blood communicated disease to *one out of five* who submitted to the experiment.

The recognition of this fact explains many occasional cases of syphilitic affection hitherto obscure—*e. g.*, syphilis from vaccination, contamination of a healthy nurse from the sore mouth of an infected infant, and the like. One of the most remarkable and lamentable instances of the inoculation of syphilis through vaccination is that which is now well known as the epidemic at Rivalta. At that place no fewer than *forty-six* children became affected with syphilis, the disease being communicated to each of them through the operation for vaccination (PACHIOLLI, SPERINO, *New Syden. Society Year Book*, 1861–62).

4. Morbid Anatomy of the Secondary Lesions and Local Growths in the Internal Viscera.—These are now so uniformly found associated with a history of syphilis that they are rightly regarded as the remote effects of the specific venereal or syphilitic virus. So varied are the effects of syphilis that a complete account of syphilis and its lesions has yet to be written; but it may be useful to illustrate some of the points of view from which the subject in its pathological bearings is now being examined, premising that it is necessary to *examine* the subject carefully from year to year, as opportunity offers and as fresh facts add to our knowledge, being ever alive to the fallacies which inevitably surround the most patient investigations.

[Syphilis in its ultimate form is capable of affecting every organ of the body; the internal organs may become equally as obnoxious to the effects of the virus as the external. Many obscure and intractable organic disorders are cases of visceral syphilis; and it cannot be too forcibly impressed upon the young practitioner, that syphilis may affect, “not only the cranium, but the brain within it, or the nerves; not only the muscles of the limbs and tongue, but the heart; not only the pharynx, but the œsophagus; not only the larynx, but the trachea, bronchi, and lungs; also the liver, spleen, and other viscera.” (S. WILKS, *Guy's Hospital Reports*, vol. ix, 1863).]

From any one of the sources of infection already noticed, the later stages of syphilis are characterized by lesions which are distinguished from the earlier specific affections, both by their situation and by their morbid anatomical peculiarities.

Differences in the stage of the disease have hitherto been mainly based upon the organs affected. The *primary affection* being local, the so-called *secondary affections* or *stages* were considered to be those which involved the skin, the mucous membranes, and the iris; while the *tertiary symptoms* or *stages* were those which implicated the areolar tissue, the bones, the muscles, the liver, the brain, the heart, lungs, and the kidneys.

A division based on the *anatomical* characters of the lesions seems to be more satisfactory than any arbitrary arrangement into stages of a supposed primary, secondary, or tertiary order (HALDANE).

1. In the so-called primary and secondary affections we have mainly to do with congestions, inflammations, and ulcers.

2. In the tertiary lesions and advanced stages of syphilis there is—(a.) A “constitutional cachexia,” with certain definite anatomical characters; and (b.) A tendency to the growth of a peculiar material, chiefly in the form of gummatous tumors or nodules, of which the *node* is the common and familiar type; but which are found not only in the bones, but in the areolar tissue, the liver, the lungs, the heart, the brain, the muscles, the testicles, the eye. (c.) There is likewise to be observed a tendency to various interstitial inflammations; and (d.) The occurrence of cicatrix-like losses of substance visible on the surface of solid organs.

With regard to the “constitutional cachexia,” it is necessary, if possible, to distinguish the degenerate nutrition brought about by “inherited” syphilis, as contrasted with that brought about by acquired syphilis.

The constitution of the person also materially influences the phenomena which supervene during syphilis—*e. g.*, gout, rheumatism, tuberculosis, and cancer modify the syphilitic lesions and degenerations; while constitutional syphilis in its turn modifies the character of ordinary diseases.

Persons with a tendency to rheumatism are apt to have the same tissues involved in syphilitic lesions as if they suffered from rheumatic inflammation. Hence syphilis is often set down as a cause of rheumatism. The serous, fibro-serous, white connective tissues are the sites of the lesion in the forms of *periostitis*, *iritis*, *corneitis*, and affections of the true skin.

In tuberculous patients those tissues are apt to be involved in the syphilitic lesions which are most prone to ulcerate, and to have tubercles grow in them. Hence syphilis is often set down as a cause of phthisis. The mucous membranes are most prone to suffer in such cases. Hence syphilitic growths develop themselves in the lungs, the glands and brain, pharynx, larynx, tonsils, tongue, and testicles.

In the gouty or vascular subjects the arterial or vascular structures and joints are apt to suffer most from the syphilitic virus, and the lesions are chiefly in the form of degenerations. Hence syphilis may be set down as a cause of disease in the great bloodvessels, leading to thoracic and abdominal aneurisms at an early period of life; and of the smaller bloodvessels, leading to *lardaceous degeneration*, or waxy degeneration of the liver, kidney, spleen, and intestines.

The lesions in syphilis eventually assume a variety of anatomical forms, but in the first instance they are to be recognized in the typical forms of *nodes*, *gummata*, *tubercles*, or *knots*, as *periostitis* and *inflammations of fibrous tissues*, tending to *caries*, *necrosis*, or *abscess*, or to *hypertrophy*, as in *exostosis*, and ultimately to *cicatrices* in various organs. Secondly, in degenerations, such as the *lardaceous*. (See page 124.)

Gummata* are the characteristic lesions of tertiary syphilis. They

* [The term gummy or gummatous, used to designate syphilitic deposits, and borrowed from the French and German writers, is an unfortunate one, suggesting a soft or semifluid tumor, whilst they are hard, and fibro-plastic.—EDITOR.]

form growths which lead to the development of elastic tumors, composed of a well-defined tissue, but with elements extremely minute. The gummatous tumor takes origin from the elements of connective tissue, or the analogues of such tissue, and hence the universality of the site of these lesions. They are like pus or an abscess in this respect.

When they first attract attention (as a node on the skin, or on the shin) they are small, solid, pale swellings, like a hard kernel, varying in size from that of a pea to the size of a haricot bean. They may be generally first seen in true skin, or subcutaneous or submucous tissue; and where the tissue is lax they grow to a considerable size, and give a sensation to the hand as if filled with gum. Repeated examinations of this growth show that in the gelatinous condition it arises from a proliferation of nuclei in the cells of the connective tissue—like the formation of granulations in a wound. The component cells appear as round, oval, or oat-shaped particles, embedded in a matrix of fine connective tissue, of a granular character, tending to fibrillation. The cells are a little larger than blood-globules, and contain granules in their interior when mature. In the young condition they are contained, and are seen to grow in groups, within the connective tissue corpuscles. In some respects they resemble tubercle, but differ thus in the mode of growth. How, then, do we recognize the nature of such growths? First, taking the history of the case as a guide, we are led to conclude entirely from the anatomical character of the growth; and when such lesions are seen in a case with a distinct history of syphilis, several questions suggest themselves for consideration. Is it the result of inflammation? Is it cancer or tubercle? Is it a syphilitic lesion? Are there traces of other similar lesions in the body? As a rule, *inflammation* leads to abscesses or hypertrophies of tissue or fibroid degeneration, and round all these syphilitic nodes we have such hypertrophy and degeneration, just as we have round tubercle nodules. Abscesses are easily recognized by the pus; and which, being altered by age, may still be anatomically recognized. *Cancerous* masses are recognized by the juice expressed from them. Here we have no juice; and the cell-elements seen in cancer are characterized by the diversity of their form and growth. Here the elements are uniform in appearance and size, and form growths less highly organized than cancers, which tend to infiltrate and involve neighboring textures; whereas the gummy syphilitic node remains isolated, and is usually surrounded by a dense but clear, semi-transparent, grayish, vascular, fibrous tissue, and very resistant to the finger. Thus these nodes appear sometimes as if inclosed in a kind of cyst, from which they may sometimes be enucleated.

By way of elimination or exclusion, therefore, we may thus come to recognize such growths as syphilitic—even without a history of syphilis (HALDANE). They have been recognized now and described in all the solid viscera of the body. The microscope has enabled us to study them with minuteness; but it is only their history, posi-

tion, mode of appearance, structural elements, cause, and results, which enable us to recognize their true character.

[The mode of formation of syphilitic growths is by a localized infiltration of soft and albuminoid matter into the connective tissue, and which subsequently hardens. They do not grow from a centre, as cancerous or tubercular deposits, which are, consequently, composed entirely of the new material. The structure of the part into which they are poured out is intact in the midst of the adventitious material. As a consequence of their mode of formation they are not so well-defined as other adventitious growths, and the lymph or fibre radiates into and is blended with the adjacent tissues, and hence cannot be perfectly isolated. The syphilitic deposit presents no distinguishing features from ordinary products of inflammation. When examined under the microscope it is found to contain fibro-plastic elements, small nuclei, fatty granules, and some amorphous matter.]

Development and Course of the Syphilitic Node.—(1.) Proliferation goes on, and a glue-like mucous fluid forms, constituting the inter-cell material. The tumor, if near the surface, melts, opens, and ulcerates, thereby giving evidence of active or acute constitutional disease. (2.) The tumor continues gelatinous and coherent [if in dense parts, deeply seated], as in gummata of the periosteum, scalp, brain, liver, lungs, and heart; thus giving evidence of constitutional disease, latent or inactive. (3.) Having arrived at a more or less complete degree of development, they may undergo a retrograde or fatty degeneration, which may eventually lead to its absorption; and this is a natural or spontaneous process of cure; but traces of the existence of such nodes may be left in the form of cystoid membranes, as in the brain; or of fibrous bands, or cicatrix-like loss of substance, as in the liver. In some instances the nodes undergo calcareous degeneration.

In the Bones these syphilitic lesions arrange themselves in two groups:

1. The primary characteristic growths, or various states of gummata or nodes, which advance to the formation of ulcers; and the death of parts in little necrotic sloughs or cores of dead bone, imprisoned within circles of bone. There are usually several points of attack; and numerous holes or pores, with furrows converging to the centre, where the lesions first commence, giving rise to characteristic stellate depressions. Caries and necrosis of the bone follow the eating ulcers of soft parts, such as the roof of the palate, the nasal septum, the laryngeal cartilages; or caries and necrosis may follow periostitis of the long bones. Internal caries and necrosis may also occur, beginning in the marrow of the bone, giving rise to suppurating osteo-myelitis. Most of these forms of syphilitic lesions are to be seen in the skull bones, especially in the forehead and anterior parts of the skull. They may be seen as superficial or deep exfoliations of the outer laminae. The deeper portions become dead in small circles, inclosed by new bone; or existing like the core of a carbuncle, they cannot be removed without enlarging the aperture through which they make known their existence. The cicatrix

trices which result after absorption or elimination of gummata in bone have a characteristic appearance, especially denoted by the want of growth in the centre, and activity of growth at the edges after loss of substance.

2. The virus of syphilis seems to have the power of fostering the growth of simple interstitial inflammatory products, and thus lead to *hyperostosis*, *exostosis*, and *hard nodes*.

In the *Skin* there are two groups of syphilitic lesions to be recognized,—(1.) Local growths, which generally assume the form of eruptions; (2.) Cicatrices, [and, sometimes keloid growths.] The local growths occur in the superficial layer of the corion (VIRCHOW, BARENSPRUNG); and ultimately tend to grow deeper and to affect more permanently the derma and subcutaneous tissue (A. T. THOMSON).

When such growths soften (as they tend to do when superficial), great destruction of tissue is the result. The cicatrices which follow are permanent and unseemly, and may ensue without any abrasion of surface. This is especially the case in papular and tubercular forms of syphilitic skin diseases. The growth is generally associated with effusion of fluid, which causes the hardness; absorption taking place, atrophy of tissue follows; there is a falling in of textures, accompanied by obliteration of bloodvessels, and resulting in an unseemly white scar.

The diagnosis of syphilitic eruptions may in general be arrived at,—(1.) *From the history of the case.* A chancre commencing at least two weeks after exposure to contagion, becoming indurated, and followed within six or eight weeks by induration of the lymphatic glands. (2.) *From the symptoms accompanying the eruption.* A dusky tint of the skin, rheumatic pains in the head and joints, *alopecia*, ulceration of the throat, *iritis* nodes, *gummata*, disease of the testicle. (3.) *From the eruption appearing in several forms at one time on the body—e. g.,* Condylomatous, roseolous, lichenous. (4.) *From the general coppery tint of the eruption.* Strumous inflammations have a dusky red or vinous tint, simple inflammations in a healthy person have a bright red tint, but syphilitic eruptions in the chronic stage have a characteristic coppery color. (5.) As a rule, there is an absence of itching in syphilitic eruptions. (6.) *Syphilitic eruptions tend to assume a more or less circular form.* (7.) When the eruptions of syphilis ulcerate, the ulcers are generally round, with *perpendicular edges and unhealthy bases.* More than one of these characters must guide the diagnosis, and not one only.

[*Nodes* beginning in, and for awhile limited to, the connective tissue, are frequently met with in women, rarely in men. Their common site is the lower extremities, about the knee. They begin as a tender, hard lump, which as it grows becomes empty and soft, with a deceptive sense of fluctuation. The skin is soon adherent, and of a dusky red color. Ulceration takes place, and a large core, like soaked wash-leather (HUTCHINSON), is exposed, which is very indolent. These nodes happen from four to ten years, or longer, after the primary sore; and are not generally symmetrical.

The *bursæ mucosæ* are often implicated, when the tissues about them

are affected; and, sometimes, independently. The bursa of the patella is most frequently involved.]

Affections of the Nails occur in two forms, namely,—(1.) As *onychia*; and (2.) As a dark-red eruption on the nail, due to congestion of the vascular layer beneath, with numbness, and tingling beneath the surface. The nail atrophies, desquamation from its surface commences, and continues with splitting up of the substance and pitting. Lastly, the nail crumbles down from the edges and free margin, point, or tip. It is irregular, and thickened from the under part (Psoriasis of the nail), analogous to the "Seedy toe" of horses. (Compare Richardson's very interesting paper in *Clinical Essays*, vol. i.)

In the Substance of the Heart Virchow describes the syphilitic growth, and refers to cases of a similar kind recorded by Ricord and Lebert. Ricord, in his atlas, gives illustrations of them, and calls them "Syphilitic muscular nodes in the substance of the heart" (*Clinique Iconographique*). Firm, yellow, cheese-like masses were found in the substance of the ventricles. There was a history of old chancres and ulcerated tubercles of the skin.

In Lebert's case these gummata were seen at a comparatively early stage of development, and were found in the wall of the right ventricle. There were tubercles of the skin, of the subcutaneous tissue, genital organs, and bones of the skull (*Anat. Pathologique*). In Virchow's case there were syphilitic gummata in the testicles.

Dr. Haldane records a case of a similar kind, and gives excellent drawings in *Edin. Monthly Med. Journal* for Nov., 1862.

In the Museum of the Army Medical Department at Netley there are two preparations which show such gummata in the substance of the heart. One occurred in the case of a soldier, twenty-four years of age, under treatment for venereal ulcers of nine months' duration, on various parts of the body. He had lost his palate, and eventually sunk from exhaustion, with symptoms of phthisis. Sections of the muscular substance of the heart showed several isolated deposits in its substance and beneath its serous covering; and isolated portions of the lungs were converted into a substance of the consistence of cheese.

[From a case recorded by Dr. Wilks, it is probable that the pericardium may become affected.]

In the Brain such gummatous tumors have been especially described by Bonnet, Ricord, Cullerier, and Lallemand. Ricord describes them under the name of the syphilitic tubercle of the brain. Dr. Steenberg (physician for the insane at Schleswig) believes that a great proportion of the syphilitic affections of the brain are subsequent to lesions of the arteries; and the organs of circulation generally he observes to be the frequent seat of syphilitic localizations. Hence softening of the cerebral substance, and various lesions of the nervous system, are by no means rare in cases

of prolonged syphilis; and Virchow has frequently noticed lesions of the great vessels in those who die from syphilis with lesions in the brain. The tendency to aneurismal dilatations and cicatricial-like loss of substance in the lining membrane of the great vessels, in young subjects who are severely affected with syphilis, is a subject in morbid anatomy which requires yet to be investigated.

In the cases where cerebral symptoms have long coexisted with syphilis, "a quantity of tough, yellow, fibrous tissue unites together the surface of the brain with the adjacent membrane, and this again is adherent to the bone. The cortical substance of the brain at the affected spot is often partly destroyed, and the adventitious material occupies its place. The question has still to be solved as to what structure is primarily affected. Many have given the authority of their name to the opinion that the disease commences first in the bone, but simply for the reason that the osseous system is that which has so long been recognized as liable to be affected. But since we now know that other structures may be similarly attacked, we are prepared to look for its commencement in other parts, and even in the brain-structure itself. The cases which are so frequently met with are those where the deposit involves both sides of the *dura mater*, and includes in it the bone on one side and the brain on the other. The probabilities are in favor of its occurring in the *dura mater* first, as it arises in the periosteum on the exterior of the cranium" (Wilks in *Med. Times and Gazette*, Oct. 25, 1862).

But the lesion also occurs in isolated gummatous nodules in the great nervous centres, such as the *thalami optici* or *corpora striata*. I saw very recently (29th May, 1863) a most interesting dissection of such a case in the Middlesex Hospital, which had been under the care of Dr. Goodfellow. There had been a history of syphilis, and some of the children of the man had died of inherited secondary syphilitic lesions. A gummatous tumor occupied the left *optic thalamus*. Numerous cases of syphilitic tumor of the nerves and nervous centres are to be found collected together in the pages of the *Medical Times and Gazette*, and two may be referred to of the intra-cranial nerves, related in the 17th vol., for 1858, p. 419, in each of which paralysis was due to such syphilitic *neuromata*.

The lesions of encephalic syphilis are ushered in by obscure phenomena; but the following may be especially noticed: *Insomnia* manifests itself at the commencement; and *headache* is characterized by—(1.) Violence; (2.) Prolonged duration; (3.) Nocturnal recurrence or exacerbation. The *general nervous symptoms* are especially obvious in alteration of intelligence, of sensibility, and of motion. These, combined with such obvious local lesions as caries, or necrosis of the facial bones or of the cranium; or tumors on the external surface of the cranium, such as gummata, periostitis, or exostosis, at once point to cerebral syphilitic lesions; which are sometimes expressed by persistent epilepsy.

[The disorders of the nervous system caused by syphilis are innumera-

ble, and long ago attracted attention (Von Hutten, 1519, Astruc, 1736, Van Swieten, 1773); and during the past forty years have been studied by Lallemand, Schützenberger, Virchow, Lagneau, Zambaco, Lancereau, Lacharrière, Wilks, W. Moore, Jaksch, and others. Besides neuralgia, long recognized as one of the results of syphilitic poisoning, chorea would appear to be occasionally developed under its influence (ZAMBACO). But the most common effect of the poison upon the nervous system, and the one of chief practical importance, is the production of paralysis. Mr. Hutchinson remarks, that so frequently is syphilis a cause of paralysis, that in all cases where it happens without evident cause, and in which syphilitic antecedents are even possible, it is advisable to try the effects of specific treatment. Undoubtedly we often by such means get a clue to the real nature of many an obscure affection of the nervous system. Professor Jaksch regards hemiplegia as the most frequent form of syphilitic paralysis, appearing from five or ten months to eighteen years after the primary lesion, but in most instances from three to ten years, and often very insidiously. His conclusions are based on an analysis of fifty-two cases, twelve of which were observed in his own practice. Paralysis of the muscles of the trunk was not satisfactorily made out in a single case. As a rule, the occurrence of paralysis was preceded by various other constitutional symptoms of syphilis, especially affections of the skin, mucous membranes, glands, and bones, which had either persisted to the time of occurrence of the paralysis, or were redeveloped during the hemiplegic attack, or had been already cured by the use of mercury or iodine. Among the 52 cases there were only 9 which had no treatment previous to the hemiplegia, all the rest had taken courses of mercurials or preparations of iodine, most of them repeatedly. 29 of the patients were cured of the paralysis, 6 were improved, 5 remained uncured, and 12 died, either directly from the brain-disease, or from bed-sores, or from intercurrent diseases. The pathological appearances in the brain were—in 6 instances softening in the cerebrum (sometimes with and sometimes without gummy tumors); in 1 case softening in the cerebellum; in 3 cases abscess of the brain; in 2 atrophy of the white substance. Softening of the brain, the most frequent pathological condition, may be caused in various ways. In the first place, *gummy deposits*, when they exist, may excite inflammatory action in surrounding tissues. It would be wrong, however, to suppose that this result always follows; local softenings of the brain are found in cases where no gummy tumors are present; it has been urged that the deposits may have disappeared, but Jaksch can see no proof of this, especially as an undiscovered embolus of a cerebral artery, competent to produce softening, may have been easily overlooked. A third cause of softening is suggested, viz., gummy deposits in the pia mater pressing upon the brain-cortex, and producing atrophy and induration, a fact which is borne out by observation. Abscess of the brain may also be brought about by various causes. Caries or necrosis of the cranial bones may produce suppurative inflammation of the membranes and of the brain itself. More frequently, syphilitic abscess of the brain is a consequence of inflammation of tissues surrounding a gummy deposit, or a thrombosis; or it may result directly from purulent transformation of a syphilitic deposit. Atrophy of the larger portions of the brain may be chiefly referred to *ramollissement* and obliteration of the vessels. The *diagnosis* of syphilitic hemiplegia is usually not difficult. The specific symptoms which precede usually also accompany and follow it, so that both the invasion and the development of the paralysis present peculiarities of great value for diagnostic purposes. The *prognosis* of

syphilitic hemiplegia is usually more favorable than that of other varieties; for the disease can be recognized betimes, and be judiciously treated.

Prof. Jaksch has seen twenty-five cases of syphilitic paraplegia, resembling very much locomotor ataxy. Also general paralysis, of which he makes three groups: 1. General paralysis, with mental symptoms like those of the disease commonly known by this name. 2. Loss of muscular energy, universally, without any affection of intellectual activity. 3. Palsy of all the extremities, with perfect power of the facial and ocular muscles, &c., and undisturbed mental activity. In the first form post-mortem examination shows especially either extensive softening of the cortical substance, or else atrophy of the same, with reticulated fibrous adhesions to the membranes. In some cases there was softening, with or without gummy deposits, partly in the anterior lobes, and partly in the central ganglia; in one case there was atrophy of the white substance of the hemisphere. In a case of the second variety there was softening of the pons, and thickly scattered deposits in the arachnoid of the brain and cord; in a case of the third variety there was softening of the cervical and lumbar portions of the cord, and no brain lesion. The age of the patients ranged from 23 to 58 years; only 5 were females. As a rule, the paralytic symptoms were only developed after several years' existence of constitutional syphilis. Only 5 cases were cured, and 1 improved.

It would seem that syphilitic paralysis may exist without any material lesion being revealed after death—*sine materiâ*—and that the poison may act directly on the nerve-tissue, producing molecular changes yet inscrutable, as well as by the development of intracranial or spinal growths in the osseous, fibrous, and connective tissues.*]

Any form of syphilitic infection may be followed by nervous affections, from a year old up to old age. Syphilitic brain disease generally leads to *softening* of cerebral substance surrounding the nodule; and this softening cannot be distinguished from the softening induced by any other cause. The duration of syphilitic nervous affections averages about one year; and their natural course is characterized by intermissions; and at the outset the intermissions are very distinctly expressed.

[Ricord, Zambaco, and others, mention several cases of intermittent fever in which no progress in treatment was made until the syphilitic taint was suspected, and special remedies given.]

A point in the pathology of syphilis at present assuming considerable importance, is the influence which an open suppurating sore of a secondary or tertiary kind has in removing the tendency to the localizations of lesions elsewhere, and especially in internal parts. Dr. Steenberg says, with reference to cerebral lesions, that he has seen the existence of an ulcer of a tertiary kind act as a natural issue in subduing the irritation of cerebral lesions, an entire remission of the nervous symptoms occurring while the ulcer remained open. Hence the great benefit which often follows the use of a seton in syphilitic epilepsy.

* [Des Affections Nerveuses Syphilitiques, par D. A. Zambaco, Paris, 1862.]

Mr. Henry Lee also writes that "fresh inoculations from suppurating sores during the time of their development check the activity of other lesions of the skin;" and, no doubt, of lesions elsewhere. Hence the reputed good effects of the filthy process of syphilization may in some degree be explained by these facts, as in cases where syphilization has been continued during several or many months.

The syphilitic lesions capable of affecting directly or indirectly the sense of sight are enormous and various, [giving rise to so-called amaurotic diseases]. These involve either the optic nerve itself or the constituent parts of the eye—e. g., *choroiditis*; diffuse exudation throughout [or on, or underneath], the *retina*; atrophy of the *optic nerve* and its *papilla*, with diminution in the calibre of the central vessel; circumscribed abscesses, or partial softening developed in the course of the optic nerve (VON GRAEFE).

[All the structures of the eye may become affected by the virus,—the conjunctiva, cornea, as well as the iris, the choroid, and the retina. Mr. Hutchinson has published some very interesting cases of *syphilitic interstitial keratitis*, so often met with in the inherited disease.*]

The organ of hearing may be affected with syphilis in three different ways,—disease of the bone; of the auditory nerve; and contraction of the Eustachian tube, in connection with ulceration of the pharynx (WILKS).

The motor nerves of the eye are not infrequently affected, and Mr. Hutchinson says, "Of the cases of paralysis of the fifth nerve, third, fourth, and sixth, which have come under my notice at the Ophthalmic Hospital, a large proportion has been of syphilitic origin."†

Syphilitic Lesions of the Spinal Cord undoubtedly exist, although there are as yet not many reported cases. As in the brain, the substance, membranes, and osseous covering, may be the seat of the disorder. In the substance, the lesion may be diffuse or circumscribed. An interesting case of syphilitic sclerosis of the cord is given by Lancereaux. The membranes are found thickened, adherent to each other and to the surface of the cord (ZAMBACO, WESTPHAL, VIRCHOW). Syphilitic caries of the vertebræ may subsequently extend to and involve the spinal membranes and cord.

The spinal nerves may become affected singly, or in groups. The ischiatic nerve is most frequently attacked.]

Syphilitic Lesions in the Lungs have been long ago described by Morton, Sauvage, Portal, Morgagni, and more recently by Graves, Stokes, Walshe, Wilks, Virchow, Ricord, and Munk [and Moxon].

Two forms of syphilitic lesions of the lungs are recognizable,—(1.) *Bronchitis*, or bronchial irritation at least, with fever, in many cases precedes the skin lesions, and disappears wholly or partially when this is established; and if the syphilitic eruption suddenly disappears, bronchitis may again ensue (WALSHE). The patient may thus have all the symptoms of phthisis, yet no tubercle existing in the lung. The tendency of syphilis is thus to induce

* [A Clinical Memoir on Certain Diseases of the Eye and Ear, London, 1863. London Hospital Reports, 1865.]

† [Reynold's System of Medicine, Art. Constitutional Syphilis, vol. I.]

phthisis in those especially constitutionally predisposed, and where mercury has been taken. Periostitic thickening of the clavicle and the upper ribs is apt to lead to the belief that tubercle is present, on account of the consolidation. Care is necessary in the case of young soldiers, who, having been just discharged from hospital, after the cure of an infecting chancre, may be exposed to cold and wet on guard, and so have an attack of syphilitic bronchitis induced—the probable commencement of a growth of tubercles in the lungs, or of solitary syphilitic gummata (*Annual Report of Army Med. Depart. for 1861*). (2.) Deposit in the pulmonary substance, in the form of gummata, of the same histological constitution as the well-known subcutaneous product, which has been described by M. Ricord and McCarthy as forming in the lungs, especially towards their periphery and bases. Towards the periphery they are like nodules of lobular pneumonia. They soften, and are eliminated very much like tubercle, and have at first a consistence like scirrhus. They are non-vascular (WALSHE).

[**Syphilitic Lesions of the Œsophagus, Stomach, Intestines, Rectum, and Diaphragm.**—Syphilitic ulcerations in the pharynx may extend to the Œsophagus, and cause contraction at the upper part of this tube (WILKS). M. Cullerier, several years since, published his observations of a form of enteritis, accompanied with obstinate diarrhoea, in which submucous nodular deposits not going on to ulceration, were found. It was most common in children, though he had met with it in adults. There is no doubt that syphilitic patients are liable to dysenteric symptoms, which yield to special treatment. These are, however, generally due to ulcerations of the rectum, extending high up, as far as the finger can reach, not accompanied by condylomata, happening as a late constitutional symptom, and curable by suitable treatment (HUTCHINSON, PAGET, WILKS). Murchison and Moxon have each reported a case of syphilitic formation in the diaphragm.]

Syphilitic Lesions of the Liver.—Dittrich and Gubler were the first to give an accurate description of these syphilitic deposits in the liver. Virchow has also described a peri-hepatic lesion and a simple gummy interstitial hepatitis. The former never occurs alone, but is generally associated with the latter. The hepatic substance atrophies, and the deposit contracting, is eventually absorbed, causing a cicatrix-like mark. The liver lesions are usually among the later symptoms of syphilis, and are well described by Wedl, Virchow, Wilks, and Frerichs [Loudet and Moxon. See *Syphilitic Disease of the Liver*, vol. ii, by the Editor.]

[**Syphilitic Lesions of the Spleen and Pancreas.**—The spleen is liable to the syphilitic deposit, though to a less degree perhaps than the liver. It may occur as diffuse hyperplasia, or in pale nodular patches, with cicatrix-like indentation, which latter, according to Beer, are never found in other disorders. Moxon (*History of Visceral Syphilis*, *Guy's Hosp. Rep.*, 1868) has met with sulphur-yellow nodules, of the size of peas, plentifully scattered, deep-seated, and fatty in their centre. Gummata have been found in the pancreas.

Syphilitic Lesions of the Kidneys.—These may occur in two forms,—

diffuse or circumscribed. The first has been particularly described by Beer (*Die Eingeweide Syphilis*, 1867). There is stromal and interstitial hyperplasia, with fatty degeneration. Small fatty patches scattered through the cortical substance, with interstitial hyperplasia and lardaceous degeneration of the arteries, he regards as constantly present and characteristic. The gummata are small, roundish, of a white hue, with fatty centres. In 12 out of 27 syphilitic subjects Dr. Moxon found lardaceous change, generally associated with the large white kidney, and believes that syphilis will be recognized as one of the causes of Bright's disease. Dr. Dickinson, however, contrary to the general belief, can find no proof that it is so (*Albuminuria*, 1868).]

The Syphilitic Lesions of the Testicles have been minutely examined by Virchow and Wilks [and are among the early constitutional symptoms]. Where the general substance of the testicle is affected the deposit is interstitial, and the free portion of testicle is first attacked; then the tunica albuginea thickens, and the inflammation extends along the tubes.

The middle cones of the testicle are most frequently affected, and they increase in size from proliferation of tissue elements. The interstitial tissue softens and is red, the tubes thicken, fatty epithelium becomes developed, and atrophy results. Solitary nodules may sometimes form, varying in size from a millet-seed to a cherry. They look like yellow tubercle, and are analogous to the gummata observed in the scrotum and in the substance of the tongue, which are hard and elastic, about the size of a pea, and easily overlooked. These tumors in the testicles and tongue are peculiar, in not growing from a centre, like other tumors, but rather as infiltrations of tissue (Wilks). Thus they are not perfectly circumscribed, but are found mixed up with the adjacent tissues. [The epididymis may alone be implicated.]

[**Syphilitic Lesions of the Muscles** are occasionally found amongst the most remote sequelae. They occur as well-marked, and often abruptly limited nodules, in any of the muscles of the body, but more frequently in the sterno-mastoid, the masseter, the supra- and infra-spinati, and the muscles of the forearm and leg. They are not infrequently mistaken for cancerous tumors.]

Lesions of the Tongue of a syphilitic nature are to be seen in many of these cases. They are mostly expressed by ulcerations at the base, often in such positions that the laryngoscope only can disclose them to view. Sometimes they appear as a raw, indolent, abraded-like surface, in circumscribed patches, [or nodules due to lymph-infiltration,] on the dorsum or edge of the tongue,—the aphthous exfoliation and syphilitic tubercles of Erasmus Wilson. (See Plate 3, fig. E, of his work *On Syphilis*.)

[Their site is the muscular substance. They are hard, well-defined indurations, and, often, painful; and it is, sometimes, very difficult to distinguish them from cancer, especially when they ulcerate, the surface presenting an unhealthy look.]

Hints for the Investigation and Description of Syphilitic Ulcers.—1. Ascertain as near as possible the date of contagion, keeping in view the media or vehicles of contagion, in addition to virus from a true primary chancre—namely, from ulcers in acute secondary syphilis; from the blood of patients suffering from acute secondary syphilis; from sexual intercourse, followed by conception, with a man who is at the time suffering from syphilis in some of its active secondary forms; from mixed chancres carrying the virus; from sloughing sores carrying the virus.

2. Examine the patient, keeping in view,—

(a.) That the soft, “non-infecting” sore commences almost immediately (*i. e.*, twenty-four hours to within three days after connection). It commences as a red spot, or a point, passing very soon into a pustule and a soft suppurating sore.

(b.) That the “infecting” sore does not commence before the end of the second or beginning of the third week (eighteen to twenty-four days); and if the disease has been contracted from a secondary ulceration, not before the expiration of the third or fourth week (RINECKER). A specific sore results in the form of a papule, abrasion, fissure, or crack; the formation of pus, or an ulcer discharging pus, being an accidental occurrence.

3. Examine microscopically the discharge from all syphilitic sores, keeping in view—(1.) That a “soft, non-infecting” sore discharges pus-cells; (2.) That the fluid discharged from an “infecting” sore is not pus, but a molecular debris.

4. The irritation of an “infecting” sore may cause it to discharge pus along with the “infecting fluid.” Hence “mixed chancres.”

5. The soft, purulent, non-infecting sore may be transplanted at will, and at any time, on the patient’s body. The true “infecting” sore cannot be multiplied after glandular enlargement and general infection become developed. It remains a solitary sore.

6. Look every day for cutaneous eruptions during the existence of a primary sore.

7. Examine the lymphatic glands—not only in the vicinity of the chancre, but also those in the axilla, and the neck up to the occiput. Note as to the slowness or rapidity of the enlargement, hardness or softness, tendency to suppuration, and whether painful or not.

8. From the account of syphilis and venereal sores which has been given in the preceding pages, it must appear clear that definite nomenclature must be adhered to in describing venereal or syphilitic sores.

[The Venereal Commission already referred to, from the evidence before them, came to the conclusion, that the syphilitic sore is seen under three forms: one characterized by induration throughout its entire course; one soft in its early stage, and becoming subsequently hard; and one soft through its whole course, but which, unlike the simple local sore, is followed by constitutional disorder.

The evidence, they state, is conclusive as to the impossibility of pronouncing with certainty upon the character of a sore on its first appear-

ance, *i. e.*, as to whether it will or will not be followed by constitutional symptoms. As a rule, however, the exceptions to which are rare, a soft sore, whether followed or not by a suppurating bubo, is only a local disease, and does not infect the constitution; and a hard sore, more especially if accompanied by indurated inguinal glands, does infect the constitution.

Dr. A. Fournier has recently (*Archives Générales de Médecine*, Nov. 1867) called attention to several points, which are of great importance, and may be mentioned properly under this head.

I. After a chancre has been apparently perfectly cicatrized, but with remaining induration, this may again become the seat of ulceration, and a sore be spontaneously reproduced, exactly resembling the original chancre, and limited to its site; and this process may be repeated several times. It will heal spontaneously under simple treatment, but its secretion is contagious, and capable of communicating syphilis.

II. Sometimes large masses of post-cicatricial induration soften and ulcerate at their centre, an abscess is formed, and the matter escapes through one or more small fistulous openings.

III. Occasionally indurated masses appear in the neighborhood of the original induration (*satellite indurations*) several weeks after the healing of the chancre, distinct and separate from it, but with persisting hardness. Usually they are smaller than the initial induration, but occasionally quite as large. They may subsequently become ulcerated, or be converted into mucous patches.]

Syphilization.—By this name an operation is now known which has for its object the eradication of syphilis from the system by repeated inoculations of the virus. Dr. Boeck, of Christiania, is the most persistent advocate of this mode of dealing with syphilitic cases as a remedy against constitutional affections.

It may be useful to explain here in detail the origin and doctrine of Syphilization, previous to considering the rational treatment of the disease.

In 1844 a young French physician—Auzias-Turenne—commenced a series of experiments with the view of testing John Hunter's doctrines regarding the non-communicability of syphilis to the lower animals. He succeeded at length in producing, on monkeys inoculated with chancre matter, a disease which had all the characters of a chancre. A disease was communicated to them capable of being transferred to rabbits, cats, and horses. It appears, also, that syphilis was established in these animals; and the chancres produced by inoculation became less and less in each animal, until at length a period arrived at which the virus seemed to lose all its power. No sores of any kind occurred. When a sore was established, however, in these animals, the virus was preserved, and was capable of transmission, and of re-transmission back to man. It was inoculated from a cat upon Dr. de Welz, a German physician and Professor in the University of Wurtzburg. On him it gave rise to a hard chancre, then to constitutional syphilis, demonstrating that the virus lost none of its virulence by the transference from man to animals, and from animals back to man. Nevertheless, Turenne believed that by prolonged inoculation the system became protected.

Sperino, of Turin, next took up the question. He inoculated

persons suffering from syphilis by virus from a chancre, and repeated the inoculations once or twice a week till the virus ceased to produce any effect; and when this point was reached, all other sores had healed. This naturally gave rise to the belief that, like vaccination, the system became protected; and to this process the name of Syphilization has been given.

When we consider the suffering, the long confinement, the filthy sores, and the innumerable cicatrices left, as well as the doubtful results, the uncertain state of our knowledge regarding the virus of syphilis, and the media of its conveyance, it cannot be conceded that we are warranted in sanctioning the method of treatment by syphilization. At Copenhagen, at Florence, at Turin, and other places where large hospitals exist, extensive experiments have been carried out in public; and although time and additional evidence are both wanted to learn ulterior results, nevertheless I think the facts are capable of a totally different explanation from that which has been given them, and to which I have adverted. But let me go more into detail.

Sperino's cases and Lee's experience show that during the active existence of phagedenic suppurations and continuous suppurating sores, in a patient suffering from syphilis, the phenomena of secondary infection do not advance, but the symptoms of contamination gradually wear out. Moreover, suppurations are easily established on the syphilitic. The action set up in them by repeated inoculation—the so-called syphilization—is merely a continuous suppurative action: indurated sores are not produced. The system is already contaminated; and the infecting virus will not produce any additional specific effect. Lastly, syphilis, in course of time, tends to wear itself out of the constitution. Hence the *modus operandi* of so-called syphilization may be explained, conjointly—(1.) By lapse of time; (2.) By continuous suppurations affording a drain or source of depuration to the system; (3.) From simple non-specific ulceration being sufficient to accomplish this result, as shown by the fact that the experiments on syphilization have been effected from all forms of venereal sores, discharge having been taken indiscriminately from soft as well as true infecting chancres. Moreover, the experiments recently made by Dr. Lindwurm, of Munich, clearly prove that any curative influence which the so-called process of syphilization may possess is due to the excretory action of numerous and prolonged simple ulcerations. He submitted fourteen syphilitic patients to friction with tartar-emetic ointment, without any other treatment. When the pustules from one inunction had dried up, a fresh crop was produced by a second inunction in another place; and this was repeated. The results were, in some instances, surprisingly favorable, in others less good, and in others negative. He therefore justly considers that syphilization and tartar-emetic-ointment frictions produce like results (*New Syden. Society Year-Book*, 1860, p. 325).

The process to which the name of Syphilization has been given consists of the following details:

- (1.) Matter is taken from a sore—an indurated one by preference.

(2.) A patient suffering with secondary syphilis is inoculated with it. (3.) From the pustules (*which form in about three days*) fresh inoculations are made. (4.) Every third or fourth day continue so to inoculate, always taking matter from the last pustule as long as it continues to give any result. (5.) When it ceases to give any result, new matter is to be sought for from another primary indurated ulcer, and continuous inoculation to be made as before on the sides of the person's body. (6.) When this ceases to take effect, new matter is again to be sought for and inoculated on the arms, and so on till no further inoculations will succeed. (7.) The operator is to go on inoculating so long as any new matter will produce a pustule. (8.) When no sores can be produced, the cure is considered complete; and all the symptoms of contamination from syphilis will then be found to have vanished. (9.) During this process the diet must be good and generous, no wines or spirits being allowed. The artificial ulcers are to be covered with wet cloths, and the utmost cleanliness is necessary. (10.) The mean time required to complete the cure is said to be four months (some say six months).^{*} The very length of time implies a fallacy, for by lapse of time alone the disease, in some constitutions, is known to wear itself out (GAIRDNER); but the belief in the virtue of syphilization appears to be based on a total misconception of the nature of the results obtained by the process, and on an erroneous interpretation of the facts which suggested the process. It is therefore of importance to note that the value of syphilization as a remedy may be expressed in the following summary:

1. In the experiments on syphilization all forms of the venereal poisons have been indiscriminately used.

2. The action set up by the operation is merely a continuous suppurative action. It is not alleged that the repeated inoculations produce indurated sores. Indeed, it is proven that, once the system is contaminated, the infecting virus will not produce any specific effect so long as symptoms of syphilitic contamination continue.

3. Lee's cases, and such experiments as those of Dr. Lindwurm, show that if continuous suppuration is maintained, the phenomena of secondary infection do not advance, but tend to wear themselves out; and that suppurations are easily set up in those contaminated by syphilis.

4. The *modus operandi* of syphilization is therefore explained—(a.) By lapse of time; (b.) By continuous suppurations (simple), affording a drain or source of depuration to the system.

[An Italian surgeon, Dr. Amilcare Ricordi, has recently put the subject of inoculation to the proof of observation and experiment, and his results are totally opposed to those of Drs. Boeck and Bidentkap. His object was to test the truth of those syphilographers who declared they employed the purulent secretions from the true or indurated chancres for the purpose of syphilization. If the chancre or mucous tubercle did not

^{*} [In 252 cases treated by Dr. Boeck, the average time for each case was rather more than 19 weeks (*Recherches sur la Syphilis*, p. 471.)—EDITOR.]

yield pus, it was made to do so by slight artificial irritation. He inoculated thirty-six times with pus obtained from as many hard sores, and forty-three times with that from ulcerating mucous patches, and in every case with negative results. He states that Prof. Pellizzari, in 1865, also made a similar series of experiments, and with like effects. Dr. Gustavo Bargini communicated to Dr. Ricordi the results of Bidekap's experience in Paris. He says, he made between seventy and eighty experiments with the secretions of the indurated sore, but he never succeeded in producing a characteristic ulcer (*Annali Universal. di Med.*, Jan. 1866, quoted by H. Lee, *Lancet*, April, 1866). Mr. Lee says, that in all the experiments he has performed, where due precautions have been taken, he has always failed to produce a lineal series of inoculations from an uncomplicated indurated sore; and Dr. Boeck, he adds, did not, during his residence in England, succeed in producing such a result in any one instance. Mr. Walter Coulson has had two cases in which, after repeated inoculations, he obtained well-developed pustules. There was a possible source of error in one case, but in the second none could be detected.

The following conclusions of Mr. Henry Lee may be fairly adopted:

1. That no evidence has hitherto been adduced satisfactory to the profession that the infecting form of syphilis can be inoculated upon a patient who is at the same time the subject of constitutional syphilis.
2. That both from a soft sore, and also occasionally from the surface of a hard sore, matter may be taken, which may be made to produce a number of local specific ulcerations having the characters of the soft chancre.
3. That during the continued irritation of such ulcerations, the manifestations of secondary syphilis will disappear.
4. That the treatment of syphilis in this way is tedious, and inconvenient (*The Lancet*, April, 1866).]

Treatment of Syphilis.—As in the case of other *enthetic* diseases, it is clear that if the inoculation of the syphilitic virus could be recognized in time, the site of inoculation, and with it the virus, by being destroyed, subsequent infection of the system might be prevented. But experience shows we must not conclude that even by an early destruction of the sore the occurrence of constitutional infection will be always prevented. The exact nature of a sore cannot yet be recognized at a sufficiently early date (apart from all other means of diagnosis) as to whether it will or will not prove a sore carrying a virus which will infect the system. In cases where the sore is a suppurating one, *occurring late after* exposure to infection, such a sore may be of a mixed nature, and therefore is of doubtful character, and always suspicious.

The local progress of such sores may be arrested with escharotics, if they are applied at an early period of its existence, and before contamination of the system is evinced by induration of the base of the sore. Ricord and Sigmund have found that sores destroyed by the more powerful caustics, within *from three to five days*, have not been followed by syphilitic symptoms. But these may have been cases of soft chancre, which would not infect. The only efficient caustics for this purpose are—(1.) The *strong nitric acid*; or (2.)

The *potassa cum calce* (most conveniently used in the form of small sticks).

[(3). Caustic soda, either in the stick cut to a point, and kept in contact with the sore wiped dry, until pain is felt, or, in a state of deliquescence, and applied by means of a piece of hard wood, or a glass rod, dipped in it, and brushed lightly over the part. The sore should be frequently irrigated with warm water.]

Nitrate of silver is useless, from its limited action and deficiency of penetration. If the sore threatens to slough, the parts should be wiped dry, and *nitric acid* applied, and afterwards a lotion of the *potassio-tartrate of iron*, while the same drug is given internally. *Chloride of zinc* paste ([CANQUOIN'S] FELL'S) is a useful escharotic to excite a healthy action round the periphery of a sore.

[A saturated solution of the chloride of zinc, is an excellent and easily controlled caustic. In many cases of soft ones, a solution of the salt, from twenty to fifty grains to the fluid ounce of water, will be found quite efficacious. Recently a saturated solution of carbolic acid has been recommended, the sore healing rapidly under the eschar (H. COOTE). In abrasions a concentrated solution of bichloride of mercury in alcohol is suitable, or the sulphate of copper.]

If great pain attends the local progress of the chancre, *morphia* in *liquor ammonia acetatis* is highly beneficial, [or laudanum and lead-water.]

Mercury, administered during a primary sore, seems to be one of those agents which are able to break the regular order of the manifestation of symptoms, as it does to several other diseases; although it may not seem to possess any certain prophylactic power.

There are remarkable variations in opinion as to its influence in curing syphilis. At one time discussion ran high regarding its use; and, of course, extreme statements were made on both sides, while the facts adduced never warranted the extreme conclusions.

Consequently, at one time mercury has been regarded as capable of absolutely preventing the constitutional affection; at another time it has been accused of giving to the syphilitic virus the impulse which sets up the constitutional affection. It is now quite certain, however, that mercury administered continuously to the extent of salivation, or approaching it, exerts a poisonous influence, and produces constitutional effects very similar to those produced by syphilis (GRAVES); and Hunter himself says "new diseases arise from mercury alone;" while it cannot be doubted that in cases in which mercury has been freely given, we are never certain that secondary symptoms may not supervene. Bärensprung, of Berlin, during his most extensive experience, has come to the conclusion that syphilis not only can be cured without mercury, but he avows that under its use the disease is often rendered latent for months and years, and its complete cure delayed. He is of opinion that mercury deteriorates the constitution, and favors the development of

destructive local affections. The non-mercurial treatment is slower, but surer; starvation and Zitmann's* decoction being the means he employs. He believes that the proportion of cases of constitutional syphilis to those of chancre has greatly diminished since mercurial treatment has been discontinued (*Ann. de Berlin Charité*, ix, 1, 1860; *Syden. Society Year-Book*, 1861). Herman has come to similar conclusions, from his experience in the syphilitic wards of the Vienna Infirmary. He believes that the non-mercurial treatment is much more speedy and successful than the mercurial; that no relapses occur; and that cutaneous eruption is much more frequent and severe in patients who have taken mercury. The experience of Diday is not less decided. He states that mercury cannot now be said to cure syphilis radically, so as to render all relapse impossible. Its warmest advocates do not in the present day, claim more for it than the power of delaying only the appearance of the first syphilitic manifestations, and of hastening the disappearance of certain other lesions. He imputes to it positively, and on sufficient clinical evidence, the following disadvantages: 1st. It tends to render the primary ulcer phagedenic; 2d. It tends to induce *stomatitis* and *necrosis* of the alveolar borders; 3d. It produces an acute affection of the gastro-intestinal mucous membrane, and *dyspepsia*; 4th. It brings on trembling of the extremities, apoplexy, and insanity. All of these results he has seen supervene, even when the treatment by mercury was superintended and directed by the most competent and attentive practitioners.

He does not, however, withhold mercury in every case. If the primary lesion becomes an indurated *woody* chancre, mercury is to be given. If the chancre is a doubtful one, he recommends waiting till some of the early constitutional phenomena render the nature of the case evident, and indicate the probable gravity of the syphilis with which he has to deal. He employs *iodine*, *iron*, and *quinine*, on the appearance of slight relapses, with a tonic and supporting regimen. He recommends *iodides*, to combat the *chloro-anæmia*, and to relieve the pain of tertiary ulcerations.

Numerous examples may be seen in museums, which show that the poisonous effects of mercury produce the worst lesions of the two; and, when combined with the syphilitic virus in a strumous person, the worst of all. In the extreme of syphilitic infection, it ought never to be forgotten that a specific chlorosis results from syphilis, amounting to anæmia; and that mercury will bring about a similar anæmia; while numerous instances are quoted by authors,

* Zitmann's decoction is of two kinds, the stronger and the weaker. The former is compounded as follows: R. Rad. Sarzaparillæ, ℥xij; Aquæ, ℔xxiv; *Coque per horas duas et adde* Aluminis, ℥iss.; Hydrarg. Chloridi mitis (calomel), ℥ss.; Antimonii Oxy-sulphureti, ℥j; *misce. Coque ad ½, et adde* Fol. Sennæ, ℥iij; Rad. Glycyrrhizæ, ℥iss.; Sem. Anisi, ℥ss. *Infunde per horam et cola.* The dose of this decoction is half a pint to a pint morning and evening.

The weaker decoction is compounded as follows: *Cupiat residuum* decoction fortioris et adde Radicis Sarzaparillæ, ℥ij; Aquæ, ℔xxiv; *Coque per horas duas et adde* Cort. Canellæ, Cort. Limonum, Sem. Cardamomi, ʒā ℥iij. *Infunde per horam et cola.* The dose of this decoction is one pint at intervals during the day. [Mercury is the active ingredient in this preparation.]

of the poisonous effects of mercury inducing lesions similar to those of syphilis.

Both kinds of treatment (mercurial and non-mercurial) have been extensively tried since 1816, and formal experiments have been organized on the subject, namely,—*First*, In 1822, in Sweden, by Royal command, when reports were annually furnished from civil and military hospitals as to trials of the two methods; *Second*, Dr. Fricke experimented in the Hamburg General Hospital, and published his results in 1828; *Third*, In 1833 the French Council of Health published a report on the subject.

From all these accounts more than 80,000 cases were submitted to experiment, and they go to show that syphilis is cured in a shorter time, and with less chancre of constitutional effects, by the simple than by the mercurial treatment.

It is extremely interesting and gratifying to be able to say that long before any of these reports were initiated, the surgeons of the British army perceived the ravages of the combined poisons of mercury and syphilis, and had the boldness to declare themselves against the system of treatment with mercury, and to introduce the milder measures of non-mercurial treatment.

The credit of this improvement is mainly due—(1.) To Mr. Fergusson, who practised it during the Peninsular wars (*Med.-Chir. Trans.*, vol. iv); (2.) To Mr. Rose, of the Coldstream Guards, at the same time, but independently of Mr. Fergusson; (3.) To Dr. John Thomson, the first Professor of Military Surgery; who by lectures in the University and College of Surgeons in Edinburgh, and by his published writings, was mainly influential in convincing Scotch medical men of the evil effects of mercury in venereal diseases. The inquiry, begun in 1816 by these military surgeons, requires to be reinvestigated with all the present advanced knowledge of the nature of the disease which we now possess, and with a better prospect of detecting the fallacies which surround the investigations. Under the simple treatment of those eminent men, there can be no doubt that mixed cases of soft, as well as of indurated chancres, and specific or syphilitic sores, were allowed spontaneously, as it were, to develop their distinctive characters. No confidence can now be placed in the results derived from clinical observation, where the cure of soft, suppurating, and mixed sores, gonorrhœa, vegetations, suppurating buboes, are all indiscriminately given as evidence of the cure of syphilis; and cases cannot be accepted as cured at the time they are simply discharged from present treatment, because they may seem to be progressing to a favorable termination, but not absolutely cured.

Nomenclature, as already explained (page 678), must now be specially attended to.

The present position of opinion with regard to mercury in the cure of venereal sores seems to be this, namely,—That it is a very valuable remedy in some cases of syphilis, but not in cases of soft chancre; and the difficulty is to express always the nature of the cases for which it is most suitable. Even those who believe most fully in its virtues acknowledged that in primary affections, as when

given in the treatment of the local sore, its administration will not *prevent the occurrence* of constitutional symptoms; nevertheless, the value of mercury in the cure of THE INDURATION of the true *infecting chancre* is now fully recognized. The local lesion, if it appears after the usual prolonged period of incubation, is as much a manifestation that the constitution is already affected as is the developed vesicle of *variola vaccina* a manifestation that the constitution is affected with variolous poison. Looking, also, to the nature of the virus of syphilis, as expounded in the text (especially at page 677), the *excision* of the *primary lump* or sore—the *specific induration*—as practised by Dr. Veale (*Edin. Monthly Journal*, July, 1864), and by Dr. Humphry, of Cambridge (*British Medical Journal*, August 13, 1864), is a justifiable operation; for the original sore, when it has become a “lump” (as in its state of “woody-like” induration), is an undoubted maintainer of infection and of contamination of the system. If, therefore, it can be easily and completely insulated, as when on the *prepuce*, the cure of the constitutional symptoms may be facilitated.

There are also certain forms of secondary syphilis for which the administration of mercury is unsuitable. These are the pustular eruptions, or ecthymatous states in rupia and in syphilitic anæmia. For the cure of other secondary symptoms mercury is certainly of service. If given to the extent to which I have limited its use in the text, I believe that secondary symptoms disappear more rapidly under its regulated use than by any other plan of treatment. All our treatment of syphilis rests on that evidence which must always guide the hand of the physician, namely—practical experience. That has certainly taught us that the mercurialism of John Hunter's time was an error; and that its regulated administration in cases of *syphilis* is undoubtedly beneficial, and especially during the evolution of the specific symptoms of infection. Dr. Jeffrey Marston, of the Royal Artillery, has given an admirable summary [*British Medical Journal* of Feb. 21, 1863] of the means and indications of treatment by mercury which he has found most useful. His experience shows that the system ought to be affected as slowly as possible; and there ought to be a remission of the remedy for a time as soon as that effect has been attained.

[The antidotal, curative, or specific property of mercury in syphilis may, perhaps, be not proved; but no medical man of large opportunity, free from partisanship, and who has fairly tried both the mercurial and non-mercurial practice in true syphilis, will deny that, when properly administered, mercury hastens the healing of the primary sore, abates the induration, lessens the liability to the happening of constitutional phenomena, removes these when they appear, and in many of the syphilitic sequelæ, when the dyscrasy is fairly established, with tissue contamination, it often produces marvellous results, after other remedies have failed. Hebra and Zeissl, after giving a fair trial to all the different plans of treatment—*extractum graminis* (expectant), subcutaneous injections of cold water, iodine and its preparations, syphilization—have come back to mercury (*Brit. and For. Med.-Chir. Rev.*, vol. xxxv, 1865). Mr. Hutchinson, though denying that it is an antidote, and believing that there

is but little proof that it tends, on the whole, to abridge the duration or mitigate the severity of the constitutional symptoms, admits that it lessens the duration of the primary sore. He states that during two years of his practice in the Metropolitan Free Hospital, he abstained systematically from adopting any treatment in cases of indurated chancre and its consequences; the chancre and the rash were allowed to develop themselves and to disappear spontaneously; in each the duration was evidently longer than when mercury was given; and he finally acknowledges that "the course of syphilis, on the whole, is rendered somewhat milder by early mercurial treatment" (Reynold's *System of Medicine*, vol. i, 1866).

The Venereal Commission reported unanimously in favor of mercury, as the most efficient agent yet known in the treatment of syphilis. They say, mercury cannot be deemed a specific in the ordinary acceptance of that term, as it exercises no direct influence on the poison itself, but only on its effects. In the celebrated discussion upon syphilis, which during 1867 occupied so many sittings of the Société de Chirurgie, Dr. Verneuil said: "Till some remedy is discovered, mercury is, and will remain the most powerful agent against syphilitic accidents. If it cannot be quite admitted that its action against the disease is proved, it cannot be denied that it modifies the manifestations of the disease."

The notion that mercury is capable of producing conditions of the system similar to those following syphilitic poisoning, rests on no evidence. Necrosis of the bones has been charged upon mercury, and yet repeated experiments, made on animals by accurate observers, show that this metal does not affect the bones. In mercurialism in the human subject the bones are never implicated; Dr. Wilks states that he has seen several cases of this disease in artificers in quicksilver, where the nervous system was greatly implicated, the power of locomotion lost, the mind gone, "and the whole body undergoing decay, and yet the bones had escaped" (*Guy's Hospital Reports*, 3d ser., vol. ix, 1863). Indeed, as Dr. Wilks justly remarks, that so far from mercury producing effects resembling those of syphilis, the reverse is the case, and that it is antagonistic of the characteristic condition existing in the disease. The tendency in syphilis is from the onset of the primary sore to the production of plastic albuminoid material in the tissues, while the action of mercury is to retard its deposition, and promote its absorption. In syphilis there is a formative action; in mercury a destructive one. Where a cachexy has been established and tissue degeneration is going on, the action of mercury, being in harmony with the morbid process, is harmful. In the true syphilitic caries it is useful; in destructive necrosis of the calvaria, attended by rapid disintegration, in a cachectic individual, it would be injurious, by its toxic action on the blood, producing anæmia, and tissue changes.]

As soon as the symptoms for which the mercury was given have disappeared, steel and other remedies ought to be given; and in three cases where the general health seemed to have suffered, *podophyllin* in small doses (*one-sixth* of a grain), with extract of *belladonna*, was given with marked benefit. If the system is too early brought under the influence of mercury, and the symptoms are not benefited, *chlorate of potash* in compound tincture of *cinchona* [or *muiriate of ammonia* alone, or with *chlorate of potassa*], may be given with advantage; and in strumous subjects the *bichloride of mercury*, dissolved in *ether* and added to *cod-liver oil*, is found to be most useful. Some of the more intractable forms of syphilitic *squama* are

best treated by a combination of *liquor arsenicalis*, solution of *bichloride of mercury*, in very small doses, and tincture of *sesquichloride of iron*; while the use of soap in ablution ought to be avoided (STARTIN).

[R. Hydrarg. Chlor. Corrosiv., gr. j; Potass. Iodidi, gr. xxx; Liquor. Potass. Arsenitis, ℥xxvj; Alcohol, fʒj; Ext. Sarsaparillæ, fʒiij; Aquæ Cinnam. ad fʒxij. Two tablespoonfuls three times a day after meals.]

In the administration of mercury for the cure of syphilis, *salivation*, or anything approaching to that condition, should never be induced. *Tenderness of the gums* should be the utmost physiological effect very gradually and gently brought about. The *primary sore* should be healed without mercury; but so soon as the evolution of constitutional symptoms has commenced, such as the specific induration of the sore or glands, cutaneous, scaly, tubercular, condylomatous affection, or iritis, the sooner mercurial treatment (to the extent indicated) is commenced the greater will be the benefit. But where *suppurative lesions* have been established, mercury ought to be withheld altogether, or very sparingly combined with *iodide of potassium*.

Iodide of potassium, in doses adjusted to the individual case, appears to act with rapid benefit in some of the syphilitic diseases of the interior of the cranium giving rise to extreme pain. Its administration often causes intense suffering in patients who have been treated by mercurials. Two distinct effects are produced: *first*, the compounds of mercury fixed in the body are rendered soluble and active; and, *secondly*, a form is given to them which allows of their elimination, with more or less rapidity, in a state of combination with one of the elements of the iodide; and thus the patient is subjected anew to a mercurial treatment by the compounds of mercury already present in his body (Melsens in *Brit. and For. Med.-Chir. Review*, 1853). The dose of *iodide of potassium* should at first be small—not more than *fifteen grains* in the twenty-four hours—increasing the dose, if the patient bears it well (MELSENS, GUILLOT). Its action is aided by a blister over some portion of the shaven scalp, and by having the blistered surface dressed with mercurial ointment; and, generally, it may be said that local treatment gives very valuable aid. For example, cutaneous or mucous *raised papules* remaining persistent, an ointment composed of *oxide of zinc*, *calomel*, and *simple cerate*, hastens their absorption. Eruptions of lichen, acne, and herpes are similarly benefited by the application of *oxide of zinc lotion ointment*; and if *prurigo* and *urticaria* be also present, *diacetate of lead lotion* will expedite the cure. Vesiculo-crustaceous spots will cease to reappear if the affected parts are painted for a few days with a solution of *nitrate of silver* (gr. x-xx to ʒj), [or chloride of zinc in solution, or carbolic acid, or corrosive sublimate], and *oxide of zinc lotion* applied afterwards.

In the more advanced stages of the suppurative affections, the use of pyogenic counter-irritants ought not to be neglected, such as tartar-emetic ointment. They tend to keep up just so much of a

discharge as may be consistent with the strength of the patient; and are worthy of a trial on the principle explained under *syphilization*.

In the dry forms of syphilitic cutaneous diseases, and in *chronic eczema* of the extremities, tar ointment, or an alcoholic solution of tar, is an excellent application; and the disappearance of indolent glandular swellings is greatly aided by the use of strong solutions of iodine. Superficial forms of ulceration attending the pustules of *ecthyma* are benefited by the use of solutions of *nitrate of silver* or *sulphate of copper*, and generally by caustics and local stimulants. If a sloughing condition threaten ulcerating sores, lotions of the *potassio-tartrate of iron* will generally improve their aspect. In *psoriasis palmaris*, and similarly fissured conditions of the skin *glycerine lotions* are most useful. But all these local remedies, it must be remembered, are only aids to the constitutional treatment, whether by mercury or iodide of potassium, or simply by a well-regulated hygiene. The patient, during the whole of the treatment, should be warmly clad—should be fed upon a good but plain diet—should take plenty of exercise in the open air—should use occasionally (once or twice a week) warm baths—and avoid stimulants, unless specially indicated and prescribed.

The administration of mercury, to affect the system, is best effected through the agency of the mercurial vapor bath. It is a mode of administration not liable to affect the digestion, and it permits other remedies to be given by the mouth at the same time, if they be considered necessary. It is also mild, slow, and equable in its action,—so that it is safer than many other plans.

[The continued action of the skin which the mercurial bath keeps up, when used in primary syphilis, has apparently the effect of preventing the deeper-seated structures from becoming poisoned. Mr. H. Lee, recently, in giving his maturer experience with this treatment, states very positively, that out of a large number of cases treated both in hospital and in private practice, he has seen no severe complication of the deeper tissues, where the calomel bath has been properly used for primary syphilis (*The Lancet*, vol. i, 1866, p. 393). He gives the following very favorable statistics of this method of treatment in primary and constitutional syphilis. He excludes all cases which left the hospital before they were considered to be cured; cases complicated by some other disease not venereal; or which required some operation to be performed. Sixteen men were treated for primary syphilis, and dismissed as cured in the average time, for each case, of 5 weeks and 4 days. In thirty-two women, it gave an average of a little less than 5 weeks. Seventy-eight men were treated for constitutional syphilis by the calomel vapor bath, and discharged as cured, during 426 weeks, giving an average of little more than 5 weeks and 3 days for each case. Eighty-seven women were thus treated, during 605 weeks, giving for each case an average of something less than 7 weeks. The proportion of cachectic cases was larger in the women than in the men. Taking all these figures together, we have 213 cases treated during 1278 weeks, giving an average for each case of 6 weeks; showing a much more favorable result than that of any other method of treatment of which we have reliable statistics. The calomel vapor bath has, moreover, the advantage of being applicable

where other modes of treatment, including the internal administration of mercury, have been used and failed. It may be given where the strength is much reduced, provided there is no organic visceral disease.]

The mercurial vapor bath is to be managed in either of the following ways. The first method is best adapted for the practice of a large institution; the method recommended by Mr. Lee is better suited for private practice:

"The patient is seated on a chair, and covered with an oil-cloth lined with flannel, which is supported by a proper framework. Under the chair are placed a copper bath, containing water, and a metallic plate, on which is placed from one to three drachms of the bisulphuret of mercury, or the same quantity of the gray oxide, or the binocide of this metal. From five to thirty grains of the iodide of mercury may be employed, or a scruple of the iodide, with a drachm and a half of the bisulphuret. Under the bath and plate spirit-lamps are lighted. The patient is thus exposed to the influence of three agents,—heated air, steam, and the vapor of mercury. At the end of five to ten minutes perspiration commences, which becomes excessive in ten or fifteen minutes longer. The lamps are now to be extinguished; and when the patient has become moderately cool, he is to be rubbed dry. He should then drink a cup of warm decoction of guaiacum or sarsaparilla, and repose for a short time" (LANGSTON PARKER).

Mr. Henry Lee's mode of proceeding is more simple: "A special and convenient apparatus is used [made by J. Ronchetti, 92 Fulton Street, New York], which consists of a kind of tin case, containing a spirit-lamp. In the centre, over the flame, is a small tin plate, upon which from fifteen to thirty grains of calomel is placed, while around this is a sort of saucer, filled with boiling water. The lamp having been lighted, the apparatus is placed under a common cane-bottom chair, upon which the patient sits. He is then enveloped, chair and all, in one or more double blankets, and so he remains well covered up, for about twenty minutes, when the water and mercury will be found to have disappeared."

[There are several other points about the mode of administration of the calomel vapor bath, which are particularly insisted upon by Mr. Lee. The object of depositing the vapor of calomel on the skin is, first, to excite an action there, and secondly, that some of the mercurial may be absorbed. That this should take place, it is necessary that the powder should remain on the skin, and that the skin be soft and clean. If it is greasy, or covered with dry scales, its absorbing power is lessened, and the baths may be taken without any sensible effect. To insure proper action in the skin free perspiration should first be produced by the hot air bath—the first stage of the Turkish bath—and when the skin acts freely, the patient should be rubbed down, and immediately placed in the calomel bath. The action of the bath is materially assisted by the inhalation of a certain amount of the vapor.]

The corrosive sublimate (*Hydrargyrum corrosivum sublimatum*) is perhaps the next best form of administration; and where it has to be continued over many weeks, may be given in the following form, with opium (TANNER):

R. Hydrarg. Corrosiv. Sublimat., gr. ij; Pulveris Opii, gr. v-vij; Pulveris Guaiaci, ʒss.; *misce*; Fiant Pilulæ, xvj. Once, twice, or thrice a day.

[Dr. Scarenzio has lately published (*Annale Univ. di Méd.*) the results of the treatment of constitutional syphilis by subcutaneous injections of mercury, in eight cases. There was marked improvement in eight days, after which the eventual cure was very rapid. After experimenting with various mercurial preparations, he fixed upon calomel, obtained by steam, and combined with glycerine. Dr. Lewin, of Berlin, has treated successfully more than 700 cases of syphilis by hypodermic injections of corrosive sublimate and morphia, the mean duration of treatment being fifteen days.]

With regard to *sarsaparilla* as a remedy, Sigmund, Syme, and many other acute observers, have come to the conclusion, after long and careful trials of the best *sarsaparilla*, that it does not, *per se*, exercise the slightest perceptible influence on the course and termination of syphilitic diseases. It is usually given in combination with some mercurial preparation, as in the decoction of Zitmann (page 711, note).

Preventive Treatment.—From what has been written, it must appear clear that the only chance of preventing infection, alike on the part of the male and the female, is personal cleanliness after sexual intercourse with strangers. The good that has resulted from police inspection of females, as in Paris, Brussels, and other places, has been greatly overrated; and I believe it is mainly to be attributed to the greater attention to personal cleanliness which such inspections have brought about. If a man shall have sexual intercourse with a strange woman, let him wash the penis immediately after the act, taking care to cleanse thoroughly the folds of the *prepuce*, especially near the *frænum*, and in the sulcus of the *corona glandis*, [and use a wash of sulphate of zinc, or like preparation (PARKES), or carbolic acid lotion.] If a woman shall have sexual intercourse with a strange man, let her use a syringe with hot water, to wash out the vaginal surface, taking care to cleanse thoroughly the folds of mucous membrane at the orifice of the canal and of the *labia pudenda*. Medical inspections are formal, and look useful, leading, undoubtedly, to greater personal cleanliness, and may be the means of detecting soft chancres, and so may prevent their being communicated; but the *infecting* sore, the true *syphilitic* one, can rarely be detected in a female. The real preventive remedy is the most ancient, the most simple, and the most efficient,—wash, and be clean.

[HEREDITARY SYPHILIS.

(DR. CLYMER)

Paracelsus was the first who asserted that syphilis was transmissible from the parent to the child (1536): "Est morbus fædus . . . magis hereditarius quam lepra;" and after him, it was admitted by most of the writers of the 16th century. Ambrose Paré says (*De la grosse vérole qui survient aux petits enfans*): "Souvent on voit sortir les petits enfans du sein de leur mère, ayant cette maladie, et tôt après avoir plusieurs pustules

sur leur corps." Since then most syphiliographers have treated of hereditary syphilis, though Hunter always denied it, whilst he reported two cases which were undoubtedly of inherited origin. It is, however, only within a short period that it has been understandingly studied, and its several expressions at different periods of life satisfactorily made out, particularly the morbid changes which happen in the viscera. For the precision and extension of our knowledge on this subject, we are mainly indebted to Trousseau and Lasègue, Cullerier, Gubler, Nat. Guillot, Desruelles, Diday, Bärensprung, Förster, Henri Roger, Hutchinson, and others.

In all stages of Constitutional Syphilis the taint may be transmitted to the child. The degree of severity of the inherited taint is probably in proportion to the shortness of the period which has elapsed since the presence of active symptoms. A child may inherit syphilis in a serious form from but one parent—from its father alone, or from its mother alone. When both parents are the subject of syphilis, the child is more certain to suffer severely than when only one is so. There are as yet no sufficient data to form an opinion as to whether a child is more likely to be gravely affected when its father is the source of contamination, than when it derives the disease from its mother, or the reverse. In a large proportion of the cases met with in practice, the taint is derived from the father only (HUTCHINSON). Though infection of the offspring by the father is still contested by some authors, the numerous and positive cases collected by Bärensprung, E. Vidal, and others, put beyond doubt that constitutional syphilis of the father is transmissible to the child; but when he is suffering from the sequelæ of syphilis, the so-called tertiary stage, it rarely or never is. During the period of latency, often a protracted one, when there is no apparent manifestation of the disorder, he is capable of tainting his offspring. Bärensprung reports fourteen cases where this happened, and Diday cites several observations in support of this view. Mr. Hutchinson gives several facts in his paper published in the *London Hospital Reports*, vol. ii, p. 184, and remarks (Reynold's *System of Medicine*, vol. i, p. 299): "It is very common for a man who does not himself display a single symptom of any kind, and who appears to be in perfect health, to beget a syphilitic child, the symptoms displayed by the child being usually those of the secondary class. There is no doubt that the nearer the occurrence of the primary symptoms in the parent is to the birth of the offspring, the more certain is the latter to show symptoms of a severe character, and typically secondary in stage. Instances, however, are met in which infants, born ten years after the original disease in the parent, still display first a secondary rash, with the characteristic snuffles, &c. In several instances I have known a whole family of children, born during a period of from five to ten years, display each one the characteristic and transitory rash soon after birth." If the father alone is affected at the time of procreation, it is clear that the sperm must be the vehicle of transmission; but when the mother alone is diseased, the mode of infection of the child becomes a question. Is it through the blood, or by the ovum? Though it is contended by many that the sole agent is the blood, others believe the toxic matter to exist in the ovum, and to be subsequently developed along with it. Neither theory is supported by direct proof; analogy would favor the ovular theory, for, as Lancereaux remarks: "In view of the difficulty of the inoculation of the blood, and from the great likeness of the properties and the characters of the secretion of the testicles and that of the ovaries, there is more reason to believe in the influence of the ovum than of the blood. The hereditary transmission of

sypilis we believe to be accomplished by a modified and vitiated germ; which germ is, in fact, but an anatomical element, a cell, which like all the cells of the tainted body, has undergone impregnation by the virus" (p. 657).

Syphilis may affect the fetus at an early period, is a frequent cause of its death, and of the consequent miscarriage of the mother. According to Potton, abortion happens in one-tenth of the cases where the fetus is syphilis-tainted. Förster reports 3 deaths in 26 cases; and Whitehead found 117 miscarriages in 256 women suffering from syphilis. In these cases, appreciable lesions of the skin, viscera, and serous cavities exist in the fetus. Though the influence of the syphilitic poison may destroy the fetus, this, as Mr. Hutchinson remarks, "unfortunately is far from being its constant effect. In the great majority of such conceptions, the tainted fetus is carried to its full period;" and sometimes comes into the world with evidences of the disorder, in the shape of coryza and skin disease; but this is exceptional, for, in most cases it is apparently in good health, and it is not until after an interval of days, weeks, and sometimes months, that the disease shows itself. Usually from a fortnight to two months, the characteristic snuffling and eruption happen in a tainted child. Taking 14 observations of Henri Roger, 158 of Diday, 28 by Méric, and 49 by Mayer, a total of 249 cases, in which the appearance of the first symptoms was accurately noted, we find that they appeared during the first month in 118, or nearly one-half; and before the end of the third month, in 217, or seven-eighths; this limit being exceeded in only 32. They may, however, occur at any time before the end of the first year, though very rarely after the seventh month. There is, then, a stage of latency, extending to the period of the second dentition, puberty, or even later.

The earliest and most striking symptom of inherited syphilis, is usually the coryza, which gives the popular name of "the snuffles" to the disease. It is probably due to erythema and mucous patches of the Schneiderian membrane, which is swollen, and, stopping the nostrils, hinders breathing and sucking, and may become threatening to life. The nasal passages may be obstructed, too, by crusts and plugs of half-dried secretions. The skin and lining membrane, at the entrance of the nostrils, are cracked and ulcerated. The mouth is hot, and its mucous membrane and the gums swollen. The skin is either simultaneously or very soon affected. The exanthems differ but little from those seen in the acquired form of the disorder, generally showing the same coppery tint, and crescentic outline. Mucous patches are very common about the lips, nostrils, external palpebral commissures, anus, and genital organs of both sexes; they are moist, often fissured, soon have a whitish hue, and, when situated at the mouth or anus, give a puckered look to these orifices (TROUSSEAU). When their site is the mucous membrane, they will be found as whitish patches upon the fauces, uvula, tonsils, and, more rarely, the tongue and buccal membrane. Deep-seated pustular eruptions may appear at a later period, ordinarily upon the face; occasionally upon the neck, ears, and fold of the groin. Ecthyma and pemphigus are occasionally seen in connection with hereditary syphilis, but whether directly due to the virus, or the result of the general cachexy, is doubtful. The viscera may be affected in the same way as in the inherited disorder. A syphilitic infant has commonly a wizened and shrunken countenance, with the skin of a dirty greenish-yellow hue; there is extreme anaemia; it is puny and often stunted, and presents the aspect of old age on the threshold of life. "The face," says Trousseau, "is of a peculiar bistre tint, as if it had been washed with coffee-grounds, or a weak infusion of soot;

it is not the pallor, nor yet the icteric or strawy hue, of the other cachexiæ; it hardly extends to the rest of the body. The eyelashes are not developed, or have fallen out; the eyelids are often everted, and at the external angle are fissured. In the place of the missing eyebrows, there are yellowish scaly stains, which are sometimes found about the chin and mouth" (*Clin. Méd.*, &c., vol. iii, 1865). The child is generally fretful and cryful; sleeps but little; is troubled with vomiting and diarrhœa; and very liable to serous inflammations, as pleuritis and arachnitis, which are frequent causes of death. Erysipelas and pneumonia are common intercurrent disorders, and are generally fatal. The child sometimes dies in a state of extreme marasmus.

When the syphilitic symptoms are present to any extent at the time of birth, it is rare for the infant to live beyond a few months.

In those cases of hereditary syphilis which survive the first year, all traces of the disease disappear about that time, except perhaps unusual paleness, and an expanded nasal bridge, caused by long-continued swelling of the parts within. During the period of latency there is usually excellent health, though Mr. Hutchinson asserts condylomata sometimes reappear; but there is scarcely ever a return of the cutaneous rash. The third epoch may begin at any time after the fifth year, but it is commonly delayed until at or near the period of puberty. It is characterized by the lesions known as the tertiary stage in the acquired disorder. The diagnosis of inherited syphilis, at or after the age of puberty, may sometimes be made with much certainty, or it may be surrounded with great difficulties. "Our most valuable aids," says Mr. Hutchinson, "are the evidences of past disease, more especially of the inflammations which may have occurred in infancy. A sunken bridge of nose, caused by the long-continued swelling of the nasal mucous membrane when the bones were soft, a skin marked by little pits and linear scars, especially near the angles of the mouth, the relics of an ulcerating eruption, and a protuberant forehead, consequent upon infantile arachnitis, are amongst the points which go to make up what we recognize as an heredito-syphilitic physiognomy." In a certain number of cases a characteristic dwarfed, notched, dental malformation, will give valuable aid. It is only in the permanent set that any peculiarity is noticed; the milk teeth are liable to decay, but are not pegged, or notched. It is the *upper central incisors* which are the *test-teeth*. Even in grown-up persons, whose incisors are so much worn that the notch is obliterated, the tooth has still a diagnostic form, which Mr. Dixon likens to that of a *screw-driver*, being wide at the neck, and narrow at its cutting edge; its lateral edges are also bluntly rounded off. The complexion is usually pale, or of leaden

FIG. 4.



hue; and though the taint may dwarf the body, in most cases the general growth is not hindered. Mr. Hutchinson has met with several instances of an arrest of sexual development, and Lancereaux with one. A form of phagedenic lupus has been observed; and deafness and amaurosis from nerve or cerebral disease are both far more common in the inherited form of the disease than in that which is acquired, and are usually symmetrical. "As a rule all syphilitic symptoms in the inherited disease are

symmetrical" (HUTCHINSON). Lagneau believes that epilepsy may be one of the results, and Critchett idiocy. All the visceral deposits, met with in the later stages of the acquired disorder, may be developed in the hereditary. Lancereaux has seen chronic pneumonia developed under its influence. Nodes on the long bones, hyperostosis and caries occasionally are present; and the bones of the nose and palate may be destroyed.

Several affections of the eye are of great interest in the history of hereditary syphilis, and we owe much to the intelligent observations of Mr. Hutchinson for an accurate understanding of them. It was he who first showed, that certain morbid appearances which would have been chosen as typical specimens of strumous ophthalmia, were really due, not to scrofula, but to inherited syphilis. He named the affection Chronic Interstitial Keratitis; but it is now called Syphilitic Keratitis. It never happens in acquired syphilis. The subjects of this form of corneal disease are generally from five to eighteen years of age. The phenomena of syphilitic keratitis in the acute stage are peculiar, and easily recognized. Both eyes are usually affected about the same time; though several weeks may elapse. A diffused haziness, like that of ground glass, is first noticed in the centre of the cornea; white dots appear in the midst of the haze in the substance of the tissue, and, generally, soon run together. For awhile, the vascularity of the conjunctiva and sclerotica is but little increased, but as the corneal opacity becomes marked, these tissues become reddened, and a fine plexus of vessels spreads on to the cornea itself, gradually occupying the opaque portion, and giving to the ground glass tint a red salmon hue; and its site is commonly the upper and central part of the disk, in preference to the lower part. A zone of ciliary congestion is usually well marked. There is no tendency to ulceration. The intolerance of light is great, and the patient is often for several months, when the disorder is at its height, practically blind. The vascularity of the cornea is wholly unlike that which attends granular lids, and other chronic forms of keratitis. In the latter the vessels are large and superficial, whilst in syphilitic keratitis they penetrate the cornea so deeply, and are so fine, and so closely set together, that it gives the look of tissue infiltrated with blood (DIXON). After the inflammation has passed away the cornea most often clears in a remarkable manner, but rarely regains such perfect transparency that the traces are not left, which may be detected by the experienced observer. These traces consist in a somewhat dusky and thin sclerotic in the ciliary region, and in the presence of slight clouds here and there in the corneal substance, there being no scars in its surface. The difference between these clouds and ordinary leucomata is easily recognized (HUTCHINSON). Mr. Hutchinson has rarely seen the subjects of this affection with enlarged cervical glands, or showing other evidences of scrofula, while in thirty-one out of sixty-four cases he has recorded, he got a clear history of the occurrence of syphilitic symptoms during infancy. Most frequently it is an eldest child which suffers; and females seem to be more liable to it than males.

While Lawrence (*Lectures on Surgery*, 1863) speaks of iritis as a common symptom in infantile syphilis, Holmes (*System of Surgery*, vol. iv, p. 831, 1864) says it never occurs, and Diday does not mention it as one of the affections of inherited syphilis. It is no doubt of rare occurrence, but it does happen, and the careful researches of Mr. Hutchinson have shown that it is not quite so rare as has been supposed. Dixon in ten years saw but five or six cases out of many thousand patients treated by him in the Moorfields Ophthalmic Hospital. Mr. Hutchinson thinks

it often escapes notice, from the absence of the sclerotic zone, the small amount of local symptoms it causes, coupled with the fact that infants usually keep their eyes shut. In 23 cases collected by him, the mean age of the infant was nine months and a half; the oldest was sixteen months, and the youngest six weeks; five were males, and sixteen females, and in two the sex is not given. In 11 cases both eyes were affected. The red sclerotic zone, unfailing in adult iritis, is either wanting or scarcely traceable in the syphilitic form in the infant. The lymph is sometimes scattered over the iris in small isolated granules, but more frequently flows down to the bottom of the anterior chamber, either presenting the appearance known as *hypopyon*, or massed together in a more solid nodular form. The iris becomes dusky, the pupil irregular, and sometimes clouded by the turbidity of the aqueous humor.

The *choroid* is occasionally implicated in hereditary syphilis at about the same period of life as the cornea. The ophthalmoscope shows in such cases the presence of whitish spots on its surface, slightly raised, and covered by the retinal vessels; or the appearance of cicatrices, apparently due to the absorption of these deposits. The retina is congested, and obscured by inflammation of the membrane of the vitreous body. In the first stage, that of exudation, there is lessened vision; in the second the sight improves, the spots becoming defined; the third is that of absorption. In 14 cases reported by Hutchinson, in 10 there was choroiditis (?), in 2 retinal deposits, in 1 inflammatory opacity in the vitreous body, and in 5 opacities in the crystalline lens. In 6 out of 10, the children were the eldest born living.

The stages and symptoms of Inherited Syphilis are clearly presented in the subjoined phenomenal table by Mr. Hutchinson:

PRIMARY STAGE.—The infant usually remains without symptoms for from one week to three months.

This stage has been passed through by one or both of the sufferer's parents within from a few months to twenty years of the infant's birth. The infant is usually free from all symptoms at the time of birth.

SECONDARY STAGE.—Constitutional or exanthematic.

From the age of two to four weeks to the end of the first year.

This stage is essentially transitory, and will disappear without treatment, if the child lives.

Inflammation of nasal mucous membrane causing "snuffles."

A symmetrical and usually copious eruption on the skin. Wasting; fretfulness; a peculiar odor; a withered senile aspect; inflammation of the mouth and condylomata at anus; iritis, usually symmetrical; arachnitis, and slight effusion; disease of liver (rare); nodes (very rare). The eruptions which occur differ from those of the acquired disease, chiefly in being more moist, and in preferring the thighs and genitals. These differences may in part be due to peculiarities in the skin of young infants, and to the constant irritation from urine to which the nates are liable. Dry scaly rashes are rare. Iritis is much less frequent than in the adult, but just as well characterized when it does occur.

This stage often proves fatal.

INTERMEDIATE STAGE.—Stage of latency.

The patient will probably be wholly free from active symptoms, but will show

This stage extends from the end of the first year or eighteen months to the second dentition, the time of puberty, or even very much later.

various indications of his diathesis in pallor of the skin, sunken nose, protuberant forehead, and premature loss of the upper incisor teeth. Sometimes there will be remarkable retardation of growth and general development. If second dentition have occurred, the central upper incisors will be malformed. Unlike what happens in this stage of acquired syphilis there is scarcely ever observed any tendency to recurrence of the secondary symptoms. Now and then we see condylomata at the anus returning during the first five years, but the rash of infantile syphilis having once disappeared, scarcely ever relapses. A certain degree of nasal obstruction sometimes persists, but not often.

TERTIARY STAGE.—Stage of sequelæ.

This stage may begin with the second dentition, at the time of puberty, or not till much later. Its duration is quite indefinite.

Most of the symptoms are symmetrical: Keratitis (interstitial); kerato-iritis; periosteal nodes; cerebral deafness (not infrequent); cerebral blindness (rare); disease of liver and kidneys; phagedenic or serpiginous ulcerations of skin; cellular nodes (rare). Probably not liable to transmit the disease to offspring. Protection against a new contagion incomplete. The symmetry of the symptoms is in marked contrast with what occurs in this stage of acquired disease. The paralysis of single cranial, or spinal, nerves, so common from acquired syphilis, are, it is believed, never met with in the inherited form.

Most of the inflammations tend, unless arrested by treatment, to permanent disorganization; but one (interstitial keratitis) tends to recovery even without treatment. They are much less easily influenced by treatment than those of the acquired disease.]

CHAPTER VIII.

ON THE NATURE OF THE ACUTE SPECIFIC OR GENERAL DISEASES.

THE two orders of the diseases which have now been described constitute a group of diseases sometimes termed "*acute specific*" (WALSHE) or "*general diseases*" (WOOD), because they primarily and essentially implicate the entire system; and all of them, with the exception of dysentery, diarrhoea, croup, syphilis, and hydrophobia, may be comprehended under the term "*general diseases*." Throughout their course, and from the first, they each variously modify the composition of the blood, the calorification, and the innervation of the body. Each and all of them, also, during their progress, give rise to some lesions in the textures, of a special

anatomical character, when the disease is not too rapidly fatal to allow of these pathological features to become developed, as in *yellow fever, typhoid fever, plague, cholera*. These maladies run an acute and rapid course; they are more or less pyrexial; and, in the majority of instances, the fever which accompanies them has a fixed duration. The greater number of them are contagious, or capable of being propagated from person to person, under certain conditions not yet well understood; and, lastly, all of them are produced by an extrinsic poison, either of a *miasmatic* or specifically contaminating nature, as in those of the first order, or by the implanting of a *specific virus*, as in those of the second order.

The *specificity*, so to speak, of these diseases consists in certain characters which distinguish each of them from any other disease, and in the constancy by which, from time immemorial, such characters have continued to distinguish them. Although medical opinions regarding their pathology may change, yet the essential characters of these "*acute specific diseases*" are not known to change. Each of these diseases observes a constancy and regularity of plan in the construction and development of its morbid processes (PAGET). Each of them has some essential character or characteristics by which they are severally distinguishable. The course of the febrile phenomena is found to be distinctive, the duration of the febrile state not less so, as well as the anatomical signs which distinguish the local lesions, the development of which is concurrent with the general or constitutional phenomena; and of all truths relating to the phenomena of disease, the most important are those which relate to the order of their succession. *Specificity* cannot be denied to those diseases in which, during their natural course, we find that every phenomenon is related (in a uniform manner, so far as *exact* investigation has extended) to certain phenomena that coexist with it, and to others that have preceded and will follow it. When it is found that a series of phenomena occur in (thousands, millions) x number of instances in the same order, within similarly uniform periods of time, and altogether with so much regularity that those who are instructed, on visiting a patient for the first time, can not only affirm what has gone before, but may predict what is to come after (the highest achievement of science)—it is impossible to avoid concluding that such an invariable sequence has as constant a cause. This conclusion flows from the very constitution of our nature, and is inevitable; and on our knowledge of the facts relating to such order of succession is founded every reasonable anticipation of future events, and whatever power we possess of influencing those phenomena in the management of the disease, to the advantage of our patients and the community at large. When it is found, moreover, that there are many series of these phenomena, which may be called A, B, C, D, &c., occurring in different persons, and at different times, all perfectly distinguishable, and never by any chance capable of being confounded by a properly trained person, it is impossible to avoid concluding that the causes of A, B, C, D, &c., are not identical, and must be in fact dissimilar. Moreover, mere *uniformity* in the

sequence of such phenomena as obtains in the natural course of the respective diseases is of itself enough and sufficient (to most minds) to warrant the belief that the diseases they represent are specific. This view of the specificity of each of these diseases may be held independent of the causation of them being also specific; but the term "specific," from this point of view, necessarily means that such unlike effects must have unlike causes; and the term "specific," as we use it, is derived simply from the fact that (following the analogy of natural history) the different diseases just named A, B, C, D, &c., have been considered as so many different species, preserving their individuality through all time, as the rose, the apple, the dog, the whale, or any other animal or plant preserves theirs. Like animals and plants also, such specific diseases may disappear from off the face of the earth, when they can no longer "struggle for existence" against the well-directed measures of sanitary science. These measures may eventually be capable of rendering the existence of many specific diseases an impossibility—as much so as the existence of a megatherium or even a wolf would now be an impossibility in this country.

The origin of all specific diseases, or "how their respective first contagia arose," is alike unknown. "This in Pathology is just such a question as in Physiology is 'the origin of species.' Indeed, it is hardly to be assumed as certain that these apparently two questions may not be only two phases of one. Hourly observation tells us that the *contagium* of small-pox will breed small-pox, that the *contagium* of typhus will breed typhus, that the *contagium* of syphilis will breed syphilis, and so forth; that the process is as regular as that by which dog breeds dog, and cat, cat,—as exclusive as that by which dog never breeds cat, nor cat, dog; and prospectively we are able to predict the results of certain exposures to contagion as definitively as the results of any chemical experiment. But retrospectively we have not the same sort of certainty; for we cannot always trace the parentage of a given case of small-pox or measles" (Simon, *Sixth Report on Public Health*, p. 54). The same may be said of animals: given any individual calf, cat, dog, or child, we cannot always trace its parentage.*

* "And here," says Mr. Simon, "notwithstanding the obvious difficulties of proof either way, some persons will dogmatize that there must have been an overlooked inlet for contagium, while others will dogmatize that there must have been in the patient's body an independent origination of the specific chemical change. Presuming (as may pretty confidently be presumed) that in the history of mankind there was once upon a time a first small-pox case, a first typhus case, a first syphilis case, &c., and admitting our entire ignorance as to the combination of circumstances under which those first cases respectively came into existence, we have no scientific reasons for denying that new 'spontaneous generation' of such contagia may take place. But, as regards some of the diseases, there are conclusive reasons against supposing that this is of frequent occurrence. Where we can observe isolated populations, we find very long periods elapse without any new rise of certain 'species' of disease. For instance, in 1846, the contagium of measles was imported by a sick sailor into one of the Faroe Islands, and led to an epidemic which attacked more than 6000 out of the 7782 inhabitants; sparing only the persons who previously had had the disease, and 1500 who were kept out of reach of contagion; but before that time there had not for sixty-five years been, in those islands, a single case of measles."

With regard to their causes, therefore, each of them appears to be produced by some distinct morbid agent—some morbid poison—a poison or virus which is capable of being multiplied in the body during the development of the particular disease. In this respect they are capable of self-augmentation (PAGET). No evident fresh cause is applied, and yet the disease increases (e. g., *syphilis*, *small-pox*, *vaccinia*, *glanders*, *hydrophobia*, and *malignant pustule*). The theory of each of them, expressed in the most general terms, is, that each of them depends upon a definite specific virus, which induces a morbid condition of the blood; and that, during the development and course of the disease, the system endeavors to discharge or transform in some way the peculiar morbid agents which have given rise to the symptoms, or which have multiplied in the body during the course of the affection. The whole blood then seems to be diseased, and nearly every function and sensation in the frame is impaired or disturbed from the state of health, before any local lesion is developed. Sometimes, indeed, the severest constitutional disturbances of a specific kind may coexist with the smallest local development of any specific lesion (PAGET); and Dr. Robert Williams has justly observed, and numerous examples have been noticed, in which “it may be laid down as a general law, that when a morbid poison acts with its greatest intensity, and produces its severest forms of disease, fewer traces of organic alterations of structure will be found than when the disorder has been of a milder character. Time, duration, or *chronicity*, is a peculiarly important and characteristic element in the nature of these diseases. They run a definite course; and we know of no specific remedy which will at once effect a cure and prove an antidote to the poison. The nearest approach to an antidote is that of quinine in the malarious fevers. They have all—(1.) A more or less defined period of incu-

And the statistical return to which I have already often referred (Parliamentary Paper, 1864, No. 12) contains another very striking illustration of the same sort of thing: England has 627 registration districts. During the ten years 1851–60, scarlatina, small-pox, and measles were (as usual) prevailing more or less throughout the country, producing among children under five years of age an average annual mortality of 802 per 100,000; i. e., by scarlatina 419, by small-pox 103, and by measles 280. In 626 of the registration districts there were deaths (and, for the most part, in not inconsiderable quantity) from one or more of those causes; not quite invariably from all of them; for forty-three of the 626 (thanks, no doubt, to vaccination) had not any death by small-pox, and among the forty-three districts which thus escaped mortality by small-pox, there was one which also had not even a single death by measles; but, with these exceptions, all the 626 districts had deaths from the three diseases—deaths by measles, deaths by small-pox, deaths by scarlatina. But the 627th district had an entire escape. In all the ten years it had not a single death by measles, nor a single death by small-pox, nor a single death by scarlet fever. And why? Not because of its general sanitary merits, for it had an average amount of other evidence of unhealthiness. Doubtless, the reason of its escape was that it was insular. It was the *district of the Scilly Isles*; to which it was most improbable that any febrile contagion should come from without. And its escape is an approximative proof that, at least for those ten years, no contagium of measles, nor any contagium of scarlet fever, nor any contagium of small-pox had arisen spontaneously within its limits. I may add that there were only seven districts of England in which no death from diphtheria occurred, and that, of those seven districts, the district of the Scilly Isles was one. Still, to say that a disease is contagious is not to say that it may not arise without contagion” (SIMON, *l. c.*).

bation or latency; (2.) A period of development towards the *fastigium* or acmé of the disease; (3.) A period of *defervescence*, during which the febrile phenomena abate; (4.) Etiologically they are quite distinct from one another; and, lastly, a period of convalescence.

The causes, then, of A, B, C, D, &c., being thus specifically different, it still remains to be determined what these causes are. This is now being done by the principle of exclusion chiefly—*i. e.*, that such and such an alleged cause cannot produce such and such effects. Thus it is now certain that cold and change of temperature never cause a *specific* disease such as any of those described; that mere moisture in the air does not; that such and such gases do not, and so on. The question seems now narrowed to this point,—that in the case of many specific diseases (probably in all) the cause is something quite special, and, in fact, of unknown origin. Nevertheless, there are some, such as small-pox and syphilis, concerning which organic chemistry may enable us to learn more definitely the "*active principle*" (to use a pharmaceutical phrase) by which they are propagated; just as we have learned that *strychnine* is the active principle of *nux vomica*; or that *morphine* is of *opium*; or *digitaline* of *digitalis*.

We are certain as we can be of anything that such an "*active principle*" exists in the pus or juice of certain pustules or papules in some of those specific diseases which are capable of being propagated by inoculation. "Thus the different ferments by which they severally are communicated have respective peculiarities of their own,—peculiarities which are primarily governed by the nature and anatomical relations of the morbid process in which each particular ferment originates. All of them are essentially unstable and transitory; but, while some of them tend under ordinary circumstances to undergo a rapid extinction, others of them can with comparative ease retain their power for long periods of time, and some apparently have not their full force till some time after they have left the diseased body; *e. g.*, *cholera*. Some of them associate themselves indistinguishably with one or more of the common excretions and exhalations of the body, others are separately tangible in vesicles and pustules, or at ulcerating or suppurating surfaces, and may or may not also exist in other products of the body; some of them are evolved in small quantity, others in very large quantity, or with very large natural admixture; some of them are fixed, others but very scantily volatile, others as volatile as if they were vapors; some of them operate easily on a second body by mere contact (more or less prolonged) with the outer or inner surface to which they are applied, others are not found to act unless they come into contact with accidentally abraded surfaces, or be thrust into the bodily substance by inoculation. Thus, in vaccine lymph and in the matter of chancres respectively, there is a *contagium* which we only know in a fixed form, and only as communicable by intentional or accidental inoculation. Also ophthalmia and gonorrhœa and glanders are communicable by the fixed *contagium* which their pus contains; but this *contagium* does not need inoculation to infect the mucous membrane to which it is applied; and as regards ophthalmia, there are reasons for suspecting

that to some extent the contagious pus may retain its activity when dry enough to float as dust in the air. In some forms of *milzbrand* (including, probably, the so-called 'malignant pustule,' which is the best known human form of the disease) the highly virulent fluids can, it is alleged, infect by soakage through the cuticle. In diphtheria the characteristic exudation is capable of infecting by contact; and though often the disease is communicated from person to person without any manifest transplantation of matter, it may be that in such cases particles of the decomposed false membrane are conveyed as a volatile *contagium*. Cholera and typhoid fever send forth their respective *contagia* for the most part, if not exclusively, as matter dissolved or suspended in the evacuations which pass from the patient's bowels; and probably these evacuations (which, at least in cholera, gradually develop their full infective force after their discharge from the body) can under some circumstances bring into similar contagious fermentation the excrement with which they are mingled in privies, drains, and cesspools, and can thus convert the effluvia and leakage from such sources into means of extensive secondary infection of air and water. The volatile *contagium* of whooping-cough is probably disengaged in large quantities by the air-passages, and as it forms, is sent forth with the breath. In typhus, small-pox, measles, and scarlatina the diffusion of volatile *contagium* occurs to a vast amount, probably with all exhalations from the body; and in addition to this, *contagium*, more or less fixed, collects abundantly about the patient's person and bedding; and, in a far less degree, something of the same sort probably occurs in erysipelas" (SIMON, *l. c.*). As regards the spread or modes of propagation of these diseases, each of them "has its own laws of communicability,—laws which must be properly understood if the danger of contagion is to be guarded against. The communication of some diseases (of *scabies*, for instance, and *favus*) is not by any true product of the human body, but consists in the migration of parasites, or germs of parasites, animal or vegetable, from one person's body to another,—a migration which of course the recipient may to any extent facilitate by dirty personal habits, and which, as regards some parasitic diseases, can scarcely be conceived to occur otherwise than in consequence of such habits" (SIMON). "The communication of the diseases that have been now considered takes place by that process which is distinctly called zymotic:* in the

* "Some of these expressions," says Mr. Simon, "are meant to hesitate between two particular assertions. In this respect they correspond to the uncertainty which at present prevails as to the exact nature of some or all morbid ferments. A few years ago it might have seemed permissible to describe without reserve the contagion of the zymotic diseases, as but some changing organic material of the first affected body. At present, however, reserve on that point is necessary. That the power of contagiousness is associated with such changing organic material is certain; but whether the power be *proper to the material*, or be only *contingently* its attribute, seems to require further investigation. The recent very interesting experiments of Professor Shroeder in Germany, and of M. Pasteur in France (published respectively in Wöhler and Liebig's *Annalen der Chemie*, and in the *Comptes Rendus de l'Académie des Sciences*), aim at proving, most extensively, an essential dependence of specific fermentatory and putrefactive changes on the presence, in each case respectively, of some characteristic molecular living thing; and they give it to be

first affected body, and by or with a specific chemical transformation of some of its material, there is generated or multiplied a specific *zyme*, *contagium*, or *ferment*; which, if transferred while active to a second (not accidentally insusceptible), will there, according to the common law of ferment, excite the same morbid phenomena, the same chemical changes, as those amid which itself was begotten" (SIMON, *l. c.*, p. 53).

CHAPTER IX.

PATHOLOGY OF THE DIETIC ORDER OF ZYMOTIC DISEASES.

COMBINED researches in chemistry, physiology, and pathology, during recent years, have cleared up much that was doubtful, and established certain principles on which proper scales of diet may be founded, so as to maintain the health of the body under a great variety of conditions as to labor, confinement, freedom, and exercise. Statistics have shown more decidedly now than hitherto how intimately disease and mortality are associated with the supply of food to the people. The records of the Registrar-General of England, and those of Sir William Wilde in Ireland, the evidence of the Commissioners relative to the supplies in the Crimea during the war against Russia in 1854, 1855, and 1856, and many valuable reports relative to the health of the Navy before and after their diet was changed, fully substantiate these statements.

While physicians were well aware of the intimate connection that existed in a general way between food and disease, it is due to the labors of chemists and physiologists especially that this connection has been reduced to an intelligible form, and that the principles which they have established are now put to a practical use.

understood that, if certain fermenting or putrefying organic matters tend by their contact to bring a given quiescent organic compound into chemical excitement like their own, this contagious power of theirs depends on their carrying with them those distinctive microscopical animal or vegetable forms which in each case respectively are the true agents of change. The conclusiveness of those experiments in the field to which hitherto they have been confined is still matter of the warmest scientific controversy; and while, therefore, it would be at least premature for me to insist upon them as evidence even in that field, it would be yet more premature for me to speculate on the possible results of an extension of similar researches to the pathology of zymotic diseases. But it is impossible to ignore their very important bearing in that direction. It may be that broad distinctions will have to be drawn among the diseases which I here speak of as zymotic, or at least between them and some or all of the traumatic infections. Indeed, I gather from Canstatt's last *Jahresbericht* that already M. Monoyer, of Strasburg, in a recent work on Fermentation, has attempted a beginning in this matter. As connected with the argument in my text, it may be worth while to mention here that the different sorts of vibriones, which M. Pasteur describes as the essential powers in putrefaction, are, according to him, not only independent of atmospheric air, but are killed by it; so that, for instance, when butyric acid is forming in a saccharine solution, the butyric fermentation may at will be stopped by a current of atmospheric air, which kills the vibrionic ferment; though, on the other hand, a current of carbonic acid may traverse the solution for an indefinite time without affecting that infusorial life."

Effects of Food on the Animal Economy.—The great fact which recent chemical and physiological investigations have established may be expressed thus: "*That the various alimentary substances made use of by man and animals contain at least four classes of constituents, each of which performs its own assigned function in the living animal economy. If the substance contains nitrogen, it seems most fitted for the nourishment of tissue, and has been called plastic or nitrogenous; if it is deficient in nitrogen, and has an excess of carbon or hydrogen, it appears to undergo combustion in the body, and is called a non-nitrogenous or a respiratory element of food (hydro-carbons); if it is fatty in its nature, it performs the double duty of maintaining animal warmth, and of assisting in the assimilation of nitrogenous compounds; and, lastly, if it is saline in its quality, it goes to build up the solid textures of the animal frame, and aids the important work of carrying new materials into the system, and old or effete matter out of it*" (LETHEBY). Man and animals cannot maintain health if their food does not contain all of these constituents; and common instinct, with experience, tells us that these classes must be associated in due proportions, under a variety of modifying circumstances. There are undoubted *habits* of feeding which, while they appear to be dictated by common instinct, are also sanctioned by science. For example, white meat being deficient in fat, bacon is eaten with veal and with fowl; melted butter is used with fish; eggs and butter does mixed with sago, tapioca, and rice; cheese is eaten with macaroni; salads and vinegar are eaten with cold or salted meat; a vegetable is mixed with an animal diet; bread is eaten with butter, bacon with greens, pork with pease pudding, and so on. Old habits and instincts not only declare that these combinations are compatible, but Science informs us now why such combinations are demanded for the maintenance of health; and when they cannot be obtained, health is endangered, the constitution is gradually altered, temperament is modified, life is shortened, families extinguished, armies are swept from their encampments, and races of men from the face of the earth.

The experience of Dr. Christison (who has paid great attention to this subject for the last twenty-four years) has shown,—(1.) That the most successful dietaries for bodies of men, deduced from practical observation, contain carboniferous and nitrogenous food in proportion of about three of the former to one of the latter by weight. (2.) That while *nitrogenous* may replace *carboniferous* food for supporting respiration, *though at a great loss, carboniferous* food (without nitrogen) cannot replace *nitrogenous* food for repairing textural waste. (3.) The daily amount of nutritive principles of both sets must increase with exercise and exposure, otherwise the body quickly loses weight, and ere long becomes diseased. If the above proportion between the two sets be maintained, the weight of real nutriment per day varies, for adults at an active age, between seventeen and thirty-six ounces; the former being enough for prisoners confined for short terms, the latter being required for keeping up the athletic constitution, or that which is capable of great continuous muscular efforts. (4.) Dietaries ought never to

be estimated by the rough weight of their constituents, without distinct reference to the real nutriment in these, as determined by physiological and chemical inquiry.

[A man of average size and activity will, under ordinary conditions of moderate work, take in twenty-four hours from $\frac{1}{10}$ th to $\frac{1}{6}$ th of his own weight in solid and liquid food; of so-called *solid food* (bread, meat, &c.), about 40 ounces (*i. e.* with an average range of from 34 to 46 ounces), and of *water* from 50 to 80 ounces; making in all from 80 to 120 ounces by weight of ingesta. The rate of the solid to the liquid food greatly varies; in most cases it is 1 to 2, but in some only 1 to 1. Much bodily activity requires a large increase in the solid, but less in the liquid food. But as the so-called solid food contains a certain percentage of water, if we consider only the water-free food, the average amount in the twenty-four-hours for healthy men will be, *water-free food* 22 to 23 ounces, *water* 60 to 90 ounces. The relative amount of the water-free food to water is usually as 1 to 4 or 1 to 5. Assuming the average to be 23 water-free ounces daily, and the mean weight to be 150 lbs., the body receives $\frac{1}{18}$ th of its own weight in water-free solids. The range in different persons is $\frac{1}{80}$ th to $\frac{1}{12}$ th of the body weight. Each pound weight of the body receives about 0.15 ounces (range from 0.1 to 0.2 ounces) of water-free food, and 0.5 ounces by weight of water in twenty-four hours, the amount differing in rest and activity. The following table gives the average amount for men of mean height (5 feet 6 to 5 feet 10) and weight (140 to 160 lbs.) under different conditions of activity.

A man will take on an average in twenty-four hours,—

	Water-free food in ounces avoird.	Water in ounces.
When nearly at rest,	18.5	70 to 90
When in moderate and usual exercise,	23	70 to 90
Under great exertion,	26 to 30	80 to 100 or more
Undergoing enormous exertion,	{ 30 to 36 or even 40 }	Uncertain.

Much depends on the kind and digestibility of the food; a larger quantity of indigestible food being taken, much passing undigested by the bowels. Of the water about $\frac{2}{3}$ ths or $\frac{1}{2}$ ths is taken as water; the rest is water in the so-called solid food. A man's food must be increased in proportion to the work; if not, one of two results follows: his usefulness as an agent of force lessens, being unable to do all his work; or he continues his work at the expense of his tissues, his weight decreases, and morbid causes act upon him more easily.

The phenomena of nutrition are owing to the various chemical interchanges of nitrogen and carbon, with the concurrent influences of oxygen and hydrogen (chiefly though not entirely in the form of water) and of various salts. A man of mean weight, height, and activity, requires in twenty-four hours,—

Nitrogen, about	250 to 350 grains.
Carbon, "	3500 to 5000 grains.
Salts, not fixed,	400

According to Ranke's experiments, the relative amount of nitrogen and carbon during rest and during activity was 1 N to 11 C, and 1 N to 15 C. On an average about $\frac{2}{3}$ ths of the carbon are given in the starches and fats, and $\frac{1}{3}$ th in the albuminates. When we speak of nitrogenous

and carboniferous food the proportions come out differently. One part of nitrogenous food to from 3 to 6 parts of carboniferous (1 to 4 being the mean) forms the usual proportion in apparently all nations (PARKES).

The histogenetic nature of food must be determined by direct physiological investigation, which should show comparatively the different influence of aliments upon the metamorphosis of matter in the essential animal tissues.

Professor Panum, of the University of Copenhagen, recently instituted a series of experiments to ascertain *the degree of accuracy with which it is possible, by quantitative determinations of urea, to discover how much albumen an individual can actually appropriate, digest, and decompose, from an indefinite quantity of food, consisting solely of albuminous matter and water.* His experiments were made on a dog, and he chose the purest albuminate which can be produced in the pure state,—the gluten of wheaten meal. The proportion between the dry albuminate taken and the urea produced was strikingly constant, being, in full feeding, 1 : 4.35 ; in medium feeding, 1 : 3.58 ; and in slight feeding, 1 : 2.81.

Professor Panum also conducted a series of experiments with a view to the solution of the practical question, *whether the production of urea can serve also as a measure of the histogenetic nutritive value of such foods as along with albuminates contain carbo-hydrates or fat in any considerable quantity?* It would appear upon the whole that 1550 grains of starch with 500 grains of butter diminished the production of urea tolerably equally by about 31 grains, corresponding to about 172 grains of dry albuminous matter.]*

Approximately, it may be concluded that a full-grown man of average weight (140 to 150 lbs.) and height (5 feet 7 inches) requires *one-twentieth* part of his weight in food during the twenty-four hours ; that is, *seven or seven and a half* pounds of food, including solids and liquids ; *one to one and a half* pounds (16 to 24 ounces) being solids, the rest water (PARKES).

On an average, it is found that a man requires *four or five* ounces of chemically dry NITROGENOUS food daily : that in a state of rest he will require *three and a half* ounces ; under a state of considerable exertion *five and a half* ounces ; and under extraordinary exertion he may require as much as *six and a half*, or even *seven* ounces of dry nitrogenous aliment daily. The quantity of HYDRO-CARBONIFEROUS aliment required to keep a man in health cannot be less than from *fourteen and a half* to *fifteen* ounces in twenty-four hours ; and even *nineteen* to *twenty-two* ounces under great exertions. The amount of FATTY MATTERS ought to equal about half the quantity of the nitrogenous aliment. But in a state of rest about *one* ounce in the twenty-four hours will be sufficient ; while under great exertion *two and a half* ounces may be required daily. The amount of WATER required varies from *seventy* to *one hundred and thirty* ounces ; and the SALTS supplied in the food should amount to from *half* an

* [For much late, interesting, and valuable matter on the constituents of food and its relation to animal heat, and muscular work, consult *On the Food of Man in Relation to his Useful Work.* By Lyon Playfair, C.B., F.R.S. Edinburgh, 1865. *Review of Panum on Food*, Br. & For. Med. Chir. Rev., July, 1866. Dr. F. Donders *On the Constituents of Food*, Dublin Med. Journal, February and May, 1866.]

ounce to an ounce daily, consisting of *chloride of sodium*, chloride of potassium, salts of *lime* and *magnesia*, carbonates, citrates, lactates, and acetates (PARKES).

TABLE I.—NUTRITIVE VALUE OF FOODS IN 100 PARTS (PARKES).

	Water.	Nitro- genous Substances.	Fat.	Carbo-hydrates, Starch, and Sugar.
Meat without Bone,*	74	16	9	...
Fat of Meat,†	68	14	14	...
Bread of average quality (White Wheaten), }	40	8	1.4	51
Starch,	100
Fat,	100	240 of Starch.
Pease,	15	24.4	2.6	49
Potatoes,	74	1.5	0.6	29
Rice,	10	3.8	0.8	85.2
Milk,	87	4.3	8.7	5.2
Maize (after Pozziate),	13.5	9.9	6.7	64.5
" (Von Bilra),	10.6	15.09	8.8	67.46

* The mean of usual statements.

† Calculated by Dr. Parkes from the statements of Lawes and Gilbert.

TABLE II.—DIETARIES AND THEIR NUTRITIVE VALUES (LETHEBY).

Diet.	WEEKLY CONSUMPTION IN OUNCES.							DAILY DITTO.		
	Bread or Biscuit.	Meat.	Potato.	Meal, &c.	Milk.	Cheese.	Butter.	Car- bonif- erous.	Nitro- genous.	Total solid Nutri- ment.
Physiological,	140	84	3.5	127	4.0	16.7
Prison Punishment, E. County and Borough Jails—	112	8.2	1.4	9.6
Under 7 days,	121	23	39.5	12.4	2.2	14.6
Not hard labor,	172	7.8	3.2	22.8	15.4	3.5	..	15.7	3.1	18.8
Hard labor,	168	14.6	63.4	27.2	41.6	1.8	..	18.2	3.5	21.7
Scotch Prisons—										
Under 3 days,	112	28	11.2	1.9	13.1
Not hard labor,	30	7.5	152	73	175	19.0	3.4	22.4
Hard labor,	76	10	176	100	175	27.0	4.5	31.5
Irish Prisons—										
Under 1 month,	56	..	192	70	70	19.5	2.9	22.4
Not hard labor,	56	..	192	60	170	20.5	3.4	23.9
Hard labor,	64	..	219	70.5	170	22.0	3.6	25.6
Military Prisons—										
Under 84 days,	56	119	210	22.2	3.8	26.0
Over 84 days,	56	168	210	27.8	4.7	32.5
Destitute Debtors,	156	16	52	22	21	16.3	3.1	19.4
Convict Prisons,	161	36	112	12	12.8	18.4	3.6	22.0
Unions (Adults),	112	15.5	51	17	34	5.2	1.1	14.2	2.6	17.0
Unions (Children),	90	14	32	..	105	..	3.5	11.1	2.3	13.4
Lunatic Asylums,	114	23	66	16	14	7	1.3*	13.2	4.0	17.2
Public Hospitals,	93	52	56	14	7	..	3.2	12.1	3.5	15.6
Army—										
Crimea,	112†	112	14.5	4.8	19.3
Home,	168	84	112	19.4	4.8	24.2
Madras,	112	112	56	4*	16.5	4.9	21.4
Bombay,	140	112	56	66*	22.2	5.6	27.8
Field (India),	168	168*	30.7	3.6	34.3
Navy,	112†	112	56	17.7	5.0	22.7
† Navigator (Crimea),	140	140	..	28	17.8	6.2	24.0
† Navigator (Home),	320	96	64	12	4	18.6	7.7	26.3
Berwickshire Laborer,	122	224	224	27.1	7.0	34.1
Yorkshire Laborer,	280	126	28	..	210	..	49	42.2	8.8	51.0

In this table only the most important articles of diet are mentioned, although the others, excepting beer, spirits, tea, and coffee, are calculated in the daily consumption. (*) are rations of rice, and (†) rations of biscuit. Gruel is calculated at the rate of two ounces meal per pint.

‡ A name given to those laborers who are employed in excavating, and such-like laborious work, chiefly connected with the construction of railways. They are also sometimes called "navvies."

To determine by calculation the amount of these different aliments, and therefore the nutritive value of a given diet, the following scale is given (Table I). It shows the mean amount of water, nitrogenous substances, fat, and carbo-hydrates—starch and sugar—which ought to be contained in 100 parts of each of the substances in common use mentioned in Table I.*

The calamities which befell our soldiers in the Crimea (in 1854) show that the dietaries of working men cannot be safely reduced below the physiological standard; and, in the words of Dr. Christison, "any person conversant with the science of the present subject could have foretold, as a certain consequence, sooner or later, of their dietary, that the British would fall into the calamitous state of health which befell them in the Crimea."

The preceding very interesting tables (II and III) of dietaries and their nutritive values, and of the nutritive values of foods, by Dr. Letheby, of London, are given here to show the actual proportions in which various substances used as food are associated in the several public dietaries of the country, and as a guide to the student of medicine when, as a practitioner, it may often be his lot to devise and construct scales of diet suited to various conditions of existence.

TABLE III.—NUTRITIVE VALUE OF FOODS (LETHEBY).

SUBSTANCES, 100 PARTS.	Water.	Fibrine, Albu- men, &c.	Starch, Sugar, &c.	Fat.	Salts.	Carbon- iferous	Nitro- genous.	Total Nutri- ment.
Human Milk,	89	3.5	4.2	3.0	0.2	11.4	3.5	14.9
Cow's Milk,	86	4.5	5.0	4.1	0.7	14.8	4.5	19.3
Skimmed Milk,	87	4.5	5.0	2.7	0.7	11.5	4.5	16.0
Buttermilk,	87	4.5	5.0	0.5	0.7	6.0	4.5	10.5
Beef and Mutton,	73	19.0	..	5.0	2.0	12.0	19.0	31.0
Veal,	77	19.0	..	1.0	0.6	2.4	19.0	21.4
Poultry,	74	21.0	..	3.0	1.2	7.2	21.0	28.2
Bacon,	20	0.8	..	70.0	1.3	168.0	0.8	168.8
Cheese (Cheddar),	36	28.0	..	30.0	4.5	72.0	29.0	101.0
" (Skimmed),	44	45.0	..	6.0	5.0	14.4	45.0	69.4
Butter,	15	83.0	2.0	199.0	..	199.0
Eggs,	74	14.0	..	10.5	1.5	25.0	14.0	39.0
White of Eggs,	78	20.0	1.6	..	20.0	20.0
Yolk of Eggs,	52	16.0	..	30.0	1.3	72.0	16.0	88.0
White Fish,	79	19.0	..	1.0	1.2	2.4	19.0	21.4
Salmon,	78	17.0	..	4.0	1.4	9.6	17.0	26.6
Eel,	90	10.0	..	8.0	1.3	19.2	10.0	29.2
Wheat Flour,	15	11.0	70.0	2.0	1.7	74.8	11.0	85.8
Barley-meal,	15	10.0	70.0	2.4	2.0	75.8	10.0	85.8
Oat-meal,	15	12.0	62.0	6.0	3.0	76.4	12.0	88.4
Rye-meal,	15	9.0	66.0	2.0	1.8	70.8	9.0	79.8
Indian-meal,	14	9.0	65.0	8.0	1.7	84.2	9.0	93.2
Rice,	14	7.0	76.0	0.3	0.3	76.7	7.0	83.7
Haricoot,	19	23.0	45.0	3.0	3.6	52.2	23.0	75.2
Peas,	13	22.0	58.0	2.0	3.0	62.8	22.0	84.8
Beans,	14	24.0	44.0	1.4	3.6	47.4	24.0	71.4
Lentils,	14	29.0	44.0	1.5	2.3	47.6	29.0	76.6
Wheat Bread,	44	9.0	49.0	1.0	2.3	51.4	9.0	60.4
Rye Bread,	48	5.0	46.0	1.0	1.4	48.4	5.3	53.7
Potatoes,	74	2.0	23.0	0.2	0.7	23.5	2.0	25.5
Green Vegetables,	86	2.0	4.0	0.5	0.7	6.0	2.0	7.0
Arrow-roots,	18	..	82.0	82.0	..	82.0

In this table the carboniferous matter is calculated as starch; 10 of fat being equal to 24 of starch.

* This table is used by Dr. Parkes in the Laboratory of the Army Medical School, and he kindly permits me to give it here.

[The amount of nitrogen, carbon, hydrogen, and salts, should not alone be considered in calculating the value of diet; the form in which these elements exist is equally important. The fats and starches are not interchangeable, and should not be confounded under the common head of carboniferous. The mean amount of the four classes of solid aliments has been calculated by Dr. Lyon Playfair, from many diets.

AMOUNT IN OUNCES (AVOIR.), AND TENTHS OF OUNCES, FOR MALE ADULTS.

	Subsistence Diet, i. e., sufficient for the me- chanical force neces- sary to carry on the internal work of the body.	Diet in quietude.	Adults in full health, but with easy work.	Adults in active work.	Adults in laborious work.
Nitrogenous substances,	2.	2.5	4.2	5.5	6.5
Fat,	0.5	1.	1.4	2.5	2.5
Starch,	12.	12.	18.7	20.	20.
Mineral matters,71	.9	...
Carbon (total),	6.7	7.4	11.16	13.7	14.3

On the Food of Man in Relation to his Useful Work. By Lyon Playfair, C.B., F.R.S.
Edinburgh, 1865.]

In cases where preserved food must be used for want of fresh food, there are difficulties to be overcome which experience and science have made to disappear in a great measure. It is known that salt meat has a tendency to engender disease, and to favor the development of disease under certain circumstances; but it is not known, either physiologically or chemically, what is the exact nutritive value of salt meat in a scientific point of view. There are three circumstances connected with its use which tend to diminish its value as an article of diet, namely,—(1.) The investigations of Liebig demonstrate that the process of salting meat is very improper, for the brine extracts the juices of the flesh, and so removes most of the important constituents. (2.) The salt meat for the navy and for soldiers in the field is always highly salted, in order to keep for two years or more in every climate; and such highly salted meat must be so thoroughly steeped in cold water, to remove the salt, before it is eatable in large quantity, that much of its most nutritive constituents must be washed out—namely, its albumen and sapid extract, called osmazome (CHRISTISON). (3.) The sarcolemma of salted meat is always hardened; and hence those foods require a long time for digestion, and frequently disagree with the stomach (LETHEBY). Few can eat a pound of salted meat daily for any length of time, even when fed on rations by no means liberal. Nitrogenous elements must therefore be added to a diet where salted meat predominates, rather than increase the amount of that substance; and this is best done by adding pease, flour, currants, raisins, and oil, butter, or lard, to the ration.

"Experience has shown," says Dr. Letheby, "that there are certain articles of food which are not particularly nourishing in themselves, but which serve some very important purposes in the animal economy. This is the case with tea and coffee: in fact, the use of a vegetable infusion, containing astringent matter and an active principle rich in nitrogen, has been almost universal among mankind from the earliest times."

"The physiological action of these beverages appears to be of a somewhat singular kind; for while they excite the brain, they calm the nervous system generally, and though they produce a state of wakefulness and activity, yet they also induce a species of languor and repose. Lehmann has ascertained by experiment that coffee greatly diminishes the wear and tear of the system; it oils the machinery, as it were, and checks the waste of friction; for those who use it find that during active exercise the destruction of tissue is prevented, and that there is less demand for food; in fact, with a maximum of work to perform, and a minimum of food to accomplish it, he will best sustain his vital power who resorts now and then to a cup of tea or coffee. Hence its value as a means of economizing food, and hence its importance to the poor laboring man." In many of our large merchant-ships the crews are engaged on the condition that coffee shall take the place of grog; and those captains who are careful of the health of the men, give them warm coffee before or after they have been aloft in cold and stormy weather—a practice which cannot be too much overvalued.

Effects of Overfeeding.—Too much respiratory food favors the development of fat, and checks the proper nutrition of the muscular tissues; hence it is that rice-feeders and potato-eaters, and those who indulge in fermented liquors, are often bloated in their appearance, become extremely fat, and are not capable of prolonged exertion. The brewer's drayman is a bad subject for the wards of an hospital; for though he usually has all the appearance of a man possessed of great muscular strength and vital endurance, yet he is not so in reality, for the muscular tissues have been encroached upon by fat, and the general power has been weakened by an undue influence of the respiratory element. Most of the animals in our menageries, from a too liberal allowance of respiratory food, die from fatty degeneration. Accumulation of the nitrogenous elements in the blood is often also a prolific source of disease, and their non-elimination (as shown in previous pages) is conducive to the propagation and development of many *miasmatic* diseases. Attention has now been drawn to the influence of such a condition in establishing the characteristic diseases of overfed convicts (LETHEBY, THOMPSON).

[Corpulence.]—This is one of the most distressing results from the effects of overfeeding, or from perverted assimilation. The adipose tissue ought to form about one-twentieth part of the weight of man, and one-sixteenth of woman. Corpulence is to be considered a disease when "it renders persons, from a difficult respiration, uneasy in themselves, and, from the inability to exercise, unfit for discharging the duties of life to

others" (CULLEN). Besides the "corporeal sufferance," the corpulent are frequently the "jest of the world," and there are those, Shakspeare tells us, who "think the worse of fat men."

Symptoms.—Besides lessened vital power and unfitness for muscular work, obese persons are short-winded and troubled with drowsiness and mental as well as bodily torpor. Lord Chesterfield thought fat and stupidity convertible terms; and Shakspeare has "fat-witted." The state of the bowels is usually natural, though often more active than in persons of healthy weight; except there is excessive accumulation in the omentum, when the "pudding in the belly" causes displacement and dilatation of the intestinal canal, when the bowels become sluggish and inactive. Dr. T. K. Chambers, from his observations of 38 persons whose weight varied from 224 to 504 pounds, came to the following conclusions: That in corpulent persons, (1.) The bony framework of the body is less massive than in the spare, indicated by the smallness of the hands and feet; (2.) The skin is usually fresh-colored, and thin; (3.) The respiratory function presents a universal and well-marked peculiarity, namely,—the volume of air they are capable of containing in, and expiring from, the chest, is considerably less than the average quantity contained and expired by healthy persons of equal height. The vital capacity is diminished, and less carbon expired.* Menstruation is said to be scanty and irregular in fat women.

Corpulence may be partial as well as general, and limited to certain organs, as the abdomen in him who carries "so many tons of oil in his belly," and the breasts of the female; in both cases, besides spoiling fair proportions, it causes personal discomfort. Though corpulence is not usually met with before adult age, there is sometimes a congenital tendency to it.

Pathogeny and Causes.—In corpulence there is a want of due balance between the reception and excretion of carbon in the system; it is supplied in greater amount than required by the tissues, or consumed in the respiration. The intestinal absorbents chiefly concerned in the osmosis of the oleaginous particles are too active (CHAMBERS). Food rich in fats and starches, as oils, fat meat, greasy gravies, sugar, the farinaceous vegetables, and fluids containing oil, as milk, or alcohol and sugar, as beer and champagne, favor the production of corpulence. The negroes on sugar plantations grow fat in crop time, from eating liberally of the cane. There is a sect of Brahmins who pride themselves on their obesity, looking on it as a sign of wealth, whose diet consists entirely of farinaceous vegetables (WADD).† An enormously fat woman on exhibition in London, in 1851, was a strict vegetarian (MOORE).‡ Fothergill stated that a rigid vegetable diet would produce fat more certainly than any other means. Bees, when fed on purified sugar, make wax (which belongs to the fats) very fast; and bears exclusively fed on bread became very fat (FLOURENS). Defective sunlight is mentioned by Chambers as one of the causes of corpulence, and an interesting case is given to support the view. Though corpulent men are generally "surfeit-swelled," they are not always either large or gross eaters, but they are invariably great water-drinkers, taking often and ample draughts. Corpulence is sometimes hereditary, though all the members of a family may not be equally affected. It is frequently developed after acute illness, or dur-

* [Gulstonian Lectures, 1850; *Renewal of Life: Lectures, Chiefly Clinical*. 2d Am. ed., 1867.]

† [Cursory Remarks on Corpulence, London, 1816.]

‡ [Corpulency, Fat or Embonpoint in Excess, London, 1860.]

ing long confinement from surgical operations, or chronic diseases, in which general nutrition is not affected.

Prognosis.—When it shows itself in early infancy and goes on increasing with age, it is incurable. In childhood and early puberty it may be checked. The later it is developed the easier it is controlled. Excessive corpulence does not betoken long life. Daniel Lambert (740 pounds), died in his fortieth year; and Bright, of Malden (616 pounds), only lived twenty-eight years.* Dancel mentions the case of a man (640 pounds), who died suddenly, suffocated with fat, at about thirty years of age. There is always in such persons a tendency to fatty degenerations of the organs.

Treatment.—By modification of the diet, we are measurably able to hinder the tendency to the formation of fat in the system, and even, to a certain extent, to lessen present amount, without otherwise doing harm to the sufferer; but the treatment must be managed with care and discretion. A neglect of the due adjustment between the fats and albuminates in the food is often followed by serious disorder of the nervous system, feeble digestion, obstinate constipation, gouty symptoms (from accumulation of the lithates), general sluggishness, and, in time, evidences of some degree of malnutrition, as the writer has several times had occasion to notice in those undergoing the so-called "Banting process." It should be borne in mind, too, that the capability for muscular work does not depend alone on the degree of development of the sarcous elements, but also on nervous action, and that fat is a nerve-nourisher, and its absence cannot fail to be felt in the nervous system; the limited endurance of prize-fighters, whose nerve-tissue is starved during "training," is well known. There exists, also, a certain relation between heat produced and muscular work, and both are derived from the chemical energy of non-nitrogenous as well as nitrogenous matters (DONDEERS).† The drift in man everywhere is toward mixed food, and with its use the best physical state is reached. Experience shows that the deprivation of the starches can be borne for a long time, if fat be given; but the deprivation of fat is ill-borne, even if starches be given. In many disorders of malnutrition, fat is found efficient as among the remedies, meeting some indication, and supplying some want. The salts, too, especially those which form carbonates in the system, are necessary to the integrity of the molecular currents, and form parts of nearly all the tissues; they exist chiefly in fresh vegetables. Hence the necessity of watchfulness and prudence in regulating the food in corpulency. When living exclusively on animal food, upon the setting in of any unpleasant symptoms, the rigorous nitrogenous diet must be modified. It should be remembered too, that mental workers do not bear abstinence, and particularly deprivation of the fats and carbohydrates, so well as body workers.

Although all corpulent persons are not huge feeders, a large number of them are; they must "leave gormandizing." The oleaginous, starchy, and saccharine articles of food should be sparingly taken, or, for a while, abstained from, under the rules already laid down. The meals should be light, and eaten at comparatively brief intervals—shortening the process

* [Shakspeare, who has given with his accustomed accuracy all the mental and physical characteristics of corpulence in that "candle mine," the "greasy knight," allows "his age some fifty, or inclining to threescore." (1st Part of King Henry 4th, Act II, Scene IV.)]

† [On the *Constituents of Food and their Relation to Muscular Work and Animal Heat*. By F. C. Donders, M.D. Translated by W. D. Moore, M.D., Dublin Quar. Jour. of Med. Science, February and May, 1866.]

of digestion, and lessening the time any mass of food shall lie in the stomach. Liquids, and especially water, should be taken in small quantities, and at the end of the meal. The amount of alcohol permitted should be very small: not that it is directly a fat-former, but, as Böcker's experiments have shown, it diminishes the excretion of carbon. Malt liquors and champagne should be strictly prohibited. The light wines of France and Germany, mixed with Vichy water, may be allowed. If tea is insisted upon, it must be used without milk or sugar; a slice of lemon, Russian fashion, may be thrown on top. Turkish baths may be often prescribed with advantage, if they are followed by a feeling of "corkiness," and not of lassitude, and there is no reason to suspect fatty degeneration of any internal organ. Warm, alkaline, and salt baths, are of use by their action on the skin, and should be aided by daily friction over the whole body. During the use of the nitrogenous diet, Dr. T. K. Chambers recommends full doses of the *liquor potassæ* daily; and Dancel, the *bicarbonate of soda*. Five to ten grains of tartaric acid are dissolved in two-thirds of a tumbler of cold water, and from thirty grains to one drachm of bicarbonate of soda are added; two-thirds of the solution to be drunk during effervescence. (*Traité Théorique et Pratique de l'Obésité (trop grand embonpoint)*, etc. Par F. Dancel, Paris, 1863.) The writer has given with advantage, as he thinks, and with a view to better tolerance of the nitrogenous diet, both phosphoric and citric acids, and lemon-juice. Bromide of ammonium has been recommended. Active exercise, in the open air and in sunlight, must not be neglected. Dancel claims that *scammony* possesses the special property of bringing on greasy dejections, fat, after its use for awhile, coming away in the stools, and sometimes in lumps. He associates, in his treatment of corpulence, along with the received dietetic rules, doses of the *tincture of pure scammony*, given before breakfast, every second or third day, in a capsule: he asserts that it is well borne by the stomach. The resin of scammony being less irritating, might be advantageously substituted. Local supporters, in the form of body and breast belts, may be worn with comfort.

Mr. Banting, the court undertaker, whose pamphlet *On Corpulence* is so widely known, was placed under the following dietary by Mr. William Harvey, a London surgeon, who, in prescribing it, followed out the already published views of Dr. Chambers and of Dancel.

Breakfast—Four or five ounces of beef, mutton, kidneys, boiled fish, bacon, or cold meat of any kind (except pork), a large cup of tea (without milk or sugar), and one ounce of dry toast. *Dinner*—Five or six ounces of any fish (except salmon or eels), any meat (except pork), any vegetables (except potatoes or rice), one ounce of dry toast, fruit out of any pudding, any kind of poultry or game, and two or three glasses of good claret, sherry, or Madeira (champagne, port, or beer forbidden). *Tea*—Two or three ounces of fresh fruit, or a rusk or two, and a cup of tea without milk or sugar. The tea may be very much enjoyed when taken in the Russian fashion—i. e., with a thick slice of lemon floating on the top instead of milk. *Supper*—Three or four ounces of meat or fish, similar to dinner, with a glass or two of claret. *Nightcap*, if required, a tumbler of grog (gin, whisky, or brandy, without sugar), or a glass or two of claret or sherry.

The quantities of the different articles specified in this liberal diet-roll, Mr. Banting states, must be left to the natural appetite, but for himself he took at *breakfast* six ounces of solid and eight of liquid food; at *dinner*, eight ounces of solid and eight of liquid; at *tea*, three ounces of

solid and eight of liquid; at *supper*, four ounces of solid and six of liquid; and the *nightcap* he introduces to show that it is not injurious; whilst, for the encouragement of smokers, it may be mentioned that tobacco is allowable.

When Mr. Banting began this treatment in August, 1862, he weighed 202 lbs., and after a year's perseverance in it, in September, 1863, he had lost 46 lbs., and had reduced his girth 12½ inches.]

Effects of Deficient Food.—"A deficiency of food, especially of the nitrogenous part, quickly leads to the breaking up of the animal frame. Plague, pestilence, and famine are associated with each other in the public mind, and the records of every country show how closely they are related. The medical history of Ireland is remarkable for the illustrations of how much mischief may be occasioned by a general deficiency of food. Always the habitat of fever, it every now and then becomes the very hotbed of its propagation and development. Let there be but a small failure in the usual imperfect supply of food, and the lurking seeds of pestilence are ready to burst into frightful activity. The famine of the present century is but a too forcible illustration of this. It fostered epidemics which had not been witnessed in this generation, and gave rise to scenes of devastation and misery which are not surpassed by the most appalling epidemic of the Middle Ages. The principal form of the scourge was known as the contagious famine fever (typhus), and it spread, not merely from end to end of the country in which it had originated, but, breaking through all boundaries, it crossed the broad ocean, and made itself painfully manifest in localities where it was previously unknown. Thousands fell under the virulence of its action, for wheresoever it came it struck down a seventh of the people, and of those whom it attacked one out of nine perished. Even those who escaped the fatal influence of it were left the miserable victims of scurvy and low fever. Another example, not less striking, of the terrible consequences of what may be truly called famine, was the condition of our troops during the early part of their sojourn in the Crimea, in 1854. With only just enough of food to maintain the integrity of the system at a time of repose, and at ordinary temperatures, they were called upon to make large muscular exertions, and to sustain the warmth of the system, in the midst of severe cold" (LETHEBY).

In cases of very gradual starvation an urgent feeling of hunger is not a prominent symptom, and even when it exists at first, it usually soon diminishes, and is succeeded by a feeling of exhaustion and faintness, and even loathing of food, if abstinence has been long protracted (R. B. HOLLAND). The mental condition connected with poverty may in part account for this deficiency of appetite. A depression produced on the nervous system is very early manifested in the impaired energies of all the vital functions, the weakened conditions of the intellectual faculties and moral feelings, and diminution of the general sensibility. Disturbance of the cerebral functions is at first shown by an unnatural languor, despondency, and listlessness, slowness and hebetude of intellect, with an inability to

employ the thoughts steadily and profitably on any subject. Notwithstanding all this general languor, however, the patient sometimes manifests a highly nervous state; he is startled by any sudden noise, and hurried by the most trifling occurrences. He is liable to attacks of giddiness, "swimming in the head," staggering, dimness of sight, with temporary delirium, and either falls as in an apoplectic fit, or lapses gradually from a lethargic state into one of stupor, or even of complete coma. In many respects the symptoms in these cases have a considerable resemblance to the effects of exposure to cold. In consequence of the torpor of the brain and intellectual faculties, it is often extremely difficult to obtain the requisite information from patients. Instead of showing any anxiety to communicate the symptoms and cause of their illness, or to relate the privations they have undergone, they generally have an unwillingness to be questioned, lie in a listless or lethargic state, without taking any notice of what is going on, and seem desirous only not to be disturbed. Such listlessness and torpor of the mental faculties, the tendency to fainting, or to perfect syncope, and, finally, a state of cerebral oppression, amounting in some cases to coma, are among the most characteristic symptoms of defective nutrition, and the surest indications of its existence to a serious extent.

[A special form of delirium, the effect of cerebral atony, is very common in prolonged inanition. (See article by editor on the Delirium of Inanition in 2d vol.) The investigations of Dr. Panum (*Virchow's Archiv*, 1864), would seem to show that, in complete inanition, though the absolute quantity of blood constantly diminishes during the progress of starvation, it is always in direct ratio to the lessening in weight of the whole body, and that the normal relative proportions of the chief constituents are maintained.]

In February of 1862 a man, thirty-six years of age, was discovered in a stack, near Morpeth, dying from starvation. All attempts to rally him failed, and he ultimately died. He was an intelligent man, and had been editor and proprietor of a penny journal called the *Falkirk Liberal*. A diary was found in his possession, containing entries of his condition from the 8th to the 25th of February; from which it appeared that during *seventeen* days he had twice tasted a piece of bread; but for the last *thirteen* days he had been entirely without food. During the first *ten* days of the *thirteen* he was able to obtain water, but on the *eleventh* day he found his legs were useless, and he lost all motor power in the lower extremities, so that "one-half of his body appeared to be dead." The case is of interest to the pathologist, as showing the length of time during which existence can be maintained if water alone be taken.

[Autophagism—the sum of the morbid manifestations resulting from prolonged abstinence from food—is a frequent intercurrent affection in both acute and chronic diseases, when their subjects have been, either from improper treatment, or from the necessity of the case, deprived of a due amount of nourishment. When a person has nothing to eat, says

Trousseau, he lives on himself. This condition is constantly met with in continued fevers, where strict diet has been enforced for any length of time, cancer of the stomach, cholera infantum, chronic diarrhœa, &c.

The quantitative estimation of the excretion of urea in a starving animal may be regarded as a pretty accurate measure of the decomposition of the essential nitrogenous tissues during inanition. Now a male adult in health excretes only about 460 grains of urea in the twenty-four hours, and if fasting, half the quantity; whilst in continued fever, pyæmia, and even acute pneumonia, the urea may rise to 775 grains, 1240 grains, and 900 or even 1075 grains, respectively, in the twenty-four hours.]

When privations of clothing and lodging are added to insufficient diet, long exertion, insufficient repose, intemperance, and the miseries of poverty, the symptoms already detailed are of the most aggravated kind. "Long before insufficiency of diet is a matter of hygienic concern—long before the physiologist would think of counting the grains of nitrogen and carbon which intervene between life and starvation—the household will have been utterly destitute of material comfort; clothing and fuel will have been even scantier than food; against inclemencies of weather there will have been no adequate protection; dwelling-space will have been stinted to the degree in which over-crowding produces or increases disease; of household utensils and furniture there will have been scarcely any,—even cleanliness will have been costly or difficult; and, if there still be self-respectful endeavors to maintain it, every such endeavor will represent additional pangs of hunger. The home, too, will be where shelter can be cheapest bought,—in quarters where commonly there is least fruit of sanitary supervision, least drainage, least scavenging, least suppression of public nuisances, least, or worst, water supply, and, if in town, least light and air. Such are the sanitary dangers to which poverty is almost certainly exposed, when it is poverty enough to imply scantiness of food" (Simon, *Sixth Report on Public Health*, 1864, p. 14). But a multitude of cases of minor degrees of suffering occur in which the symptoms are less marked than those described. Such cases are indicated by a sallow and dingy appearance of the skin, a soft and flabby feeling of the flesh, more or less emaciation, general debility, feebleness of the circulation, and frequently swelling of the ankles. The stomach becomes disordered, the appetite defective, and digestion impaired. The individual feels languid and desponding, is soon fatigued, incapable of exertion, and has an irresistible desire to fall asleep, from which he is apt to awake suddenly and in a fright. The body is easily chilled, breathlessness and palpitation are experienced after slight exertion, attacks of *vertigo*, *tinnitus aurium*, and transient blindness, are common, and there is a peculiar forlorn and dejected aspect of countenance which is very characteristic. [Aphthæ are of common occurrence in all disorders involving general malnutrition.] This state of things is commonly soon succeeded by some specific disease; though it sometimes continues, with only slight variation, for a very protracted period, until the patient falls by slow degrees into a state of mental as well

as physical incapacity; and, being no longer able to procure any employment, is completely invalided, and applies for medical relief. It may perhaps be thought that these remarks apply to cases of deficient nourishment, which are less frequent; but the experience of those who have practised extensively among the wretched purlieus and miserable abodes which exist in every large metropolitan town can testify to the contrary. I well remember listening to the interesting clinical lectures of Dr. Christison, of Edinburgh, on the cases of *scurvy* which prevailed in that town and its vicinity in 1847, and hearing the melancholy recitals of misery and starvation under which the poor suffered at that time; some under the hard taskmasters of the illegal "truck system,"* and others from absolute want at home. Among many, of whose cases I have preserved notes, a shoemaker had to support his wife and five children on *eight shillings* a week; and, to feed his children better than himself, he subjected himself to privations which in time developed *scurvy*. His daily diet consisted of *one pennynorth of bread, with tea, but no milk, in the morning—no dinner—and one pennynorth of bread, with tea, and no milk, in the evening*. After existing *three months* on this diet the disease broke out. But, apart from these extreme cases, the instances are innumerable in which deficiency of food acts as a predisposing cause of many diseases. It is now generally known that plethora and symptoms of an opposite state very nearly resemble each other, and a discrimination of these differences is of the greatest importance. While coma is often an attendant on plethora, it is not to be forgotten that it is one of the most severe and fatal signs of exhaustion from defective nutrition: and when it supervenes towards the termination of diseases of exhaustion, and the pulse becomes slower, it often acquires a degree of fulness, and gives an idea of strength, quite at variance with its previous character, and little to have been anticipated from the debilitated state of the system.

But in degrees far short of what is popularly known as starvation or famine, insufficiency of nourishment may bring very hurtful consequences to health. Local defects or local peculiarities of diet may exercise an important influence in determining or coloring particular localizations of disease; and generally it may be said, that in order justly to estimate the sanitary circumstances of a people, sufficient regard must be had to the quantity and quality of the people's meat and drink (Simon, *Report on Public Health*, p. 11, 1864).

The injurious effects produced by improper nutrition require to be studied both in relation to food and drink; and the diseases which belong to this order are *scurvy*, *purpura*, *famine fever*, *alcoholism*, and

* The "*truck system*" became developed chiefly during the formation of our great lines of railway throughout the country. The laborers (navvies) were poor, and came to work without money to buy provisions, and their field of labor was often far removed from any place where food could be bought in quantity. The contractors, their employers, then established provision stores, and in place of paying the men in money, they compelled them to take remuneration for their labor by value received in food. By this method, now declared illegal under all circumstances, the laborers often suffered from a deficient and bad supply of provisions.

probably also *rickets*. The place of *cretinism* and *bronchocele* cannot be said as yet to be definitely determined. According, however, to recent investigations, these diseases are found among people of all habits in the countries where they abound, only where the soils are composed of magnesian limestone rocks, where the waters contain an excess of magnesian salts, in France, Germany, England, Sardinia, America, and India. (See page 202.) Through the water, therefore, as an element of diet, these diseases appear to become developed, and, therefore, in the meantime, they may be arranged among the diseases of this order, some of which now demand a detailed description.

[The effects of continued insufficient alimentation have been graphically described by De Meersman, as observed in Belgium during the famine years of 1846-47 (quoted by Longet in his *Traité de Physiologie*, t. i.). The extreme emaciation of the body, pallid face, and sunken cheeks, the bright eye and dilated pupil, haggard, bewildered look, the weak, tremulous voice, the feeble memory, infirm mind, the slow, uncertain, tottering gait, dry, yellow, parchment-like, and fetid skin, stinking breath, shrunken belly, slow, sighing respiration, small, frequent, and gaseous pulse, are all described with sickening fidelity. But the largest field for the observation of the consequences of gradual continuous starvation was afforded by the Andersonville military prison towards the end of the late war, where thirty thousand men were exposed, within an area of twenty-seven acres, without shelter, and with food insufficient in quantity and quality, to the weather, with all the ills of overcrowding, and were literally, slowly and surely, starved to death. The report of Professor Joseph Jones, of Nashville, Tenn., made to the Surgeon-General of the Confederate Army, on the condition of the prisoners of war, has been well called "the most complete scientific history of inanition ever written, deduced from data which are, and probably always will be, unparalleled in magnitude." This, in a medical point of view, invaluable and instructive report, is published at length in the *Medical Memoirs of the U. S. Sanitary Commission*. (See also *Report of a Commission of Inquiry appointed by the U. S. Sanitary Commission*, Philadelphia, 1864.)]

CHAPTER X.

DETAILED DESCRIPTION OF THE DIETIC ORDER OF ZYMOTIC DISEASES.

SCURVY.

LATIN Eq., *Scorbutus*; FRENCH Eq., *Scorbut*; GERMAN Eq., *Scorbut*; ITALIAN Eq., *Scorbuto*.

Definition.—A chronic morbid state ushered in by debility, lassitude, lowness of spirits, attended by fetor of the breath, sponginess of the gums, which swell by irritation, till they overhang the teeth in palnated excrescences. Livid subcutaneous patches and spots appear upon the skin, of considerable extent, especially on the lower extremities among the roots of the hair. Spontaneous hemorrhages may take place from

the mucous canals; contractions of the muscles and tendons of the limbs occur, with pains, and sometimes superficial ulcerations. An altered state of the albumen of the blood is associated with this condition, and the phenomena are brought about by a deficient supply of the organic vegetable acids, or of the salts of fresh vegetables.

Pathology and Historical Notice.—Scurvy is mentioned by Pliny as having occurred in the Roman army commanded by Germanicus after a long encampment in Germany beyond the Rhine. It prevailed to a frightful extent in the army of Louis IX, when he was made prisoner in Egypt in 1260. With fish only for fresh provisions, dispirited by being obliged to act on the defensive, and harassed unceasingly by the Saracens, the Christian army was almost annihilated by a most terrible outbreak of scurvy. But it was not till navigation was improved, and long voyages undertaken, that this disease became well known from its general prevalence and formidable character. Vasco de Gama, in his first voyage to the East Indies by the Cape of Good Hope, in 1497, lost 100 men out of 160 by this affection. James Cartier, in his second voyage to Newfoundland, in 1535, speaks of sufferings still more severe. Of 110 people there were not ten whole. "This malady being unknown to us," he writes, "the body of one of our men was opened, to see if by any possible means the occasion of it might be discovered and the rest of us preserved. But in such sort did the malady increase, that there were not above three sound men left. Twenty-five of our best men died; and all the rest were so ill that we thought they would never recover again." A decoction of the leaves and bark of a tree, supposed to be a species of spruce fir, was the remedy which they found restored health and recovery. "It was scurvy which used to decimate our navy, and render long sea-voyages almost impossible. It was mainly by scurvy that Anson, in his celebrated voyage of 1740-2, lost within the first ten months nearly two-thirds of his crew, and during the remaining period about half of the survivors. Scurvy continued to prevail, with little abatement, till 1764. The voyages of Sir R. Hawkins, Hosier, Anson, Drake, Cavendish, Dampier, Byron, and of numerous other navigators, furnish similar details, and show how recklessly the lives of sailors were sacrificed" (COPLAND). But at last it was against scurvy that Cook had attained his triumphant success, when, in 1775, after three years' absence, he brought back a healthy crew, which, out of 112 men, had lost only one by disease. During the sixteenth, seventeenth, and earlier part of the eighteenth century, the disease was endemic* in towns, fortifications, camps, and armies. "Many thousands were often cut off within a few months in single armies and garrisons; and it is probable that more seamen perished from scurvy alone than from all other causes combined, whether sickness, tempest, or battle" (WOOD). In this country and

* It is a mistake to call scurvy *epidemic*, as has sometimes been done. The term cannot be applied with propriety to diseases other than the *miasmatic*,—diseases which we have seen to depend upon the presence of specific poisons which enter the blood by impalpable media, and common, therefore, in a greater or less extent, to all classes of the community.

in America it is now very little known, and many have never seen the disease; unless they have lived in seaport towns.

[Scurvy has always prevailed in the United States Army to a considerable extent, and under the same circumstances,—a deprivation of fresh vegetables. From 1840 to 1859, inclusive (excluding the years 1847–48—the period of the Mexican war—no reports having been made during that time), there were 4935 cases of scurvy reported and 52 deaths, in an aggregate strength of 187,144 men (*Statistical Report U. S. Army. By authority.* 1860). In the column which marched on the city of Mexico, the men, for some time previous to their landing at Vera Cruz, during the siege, and afterwards, could obtain no vegetables, and the writer is informed, on reliable authority, that on their arrival at Jalapa, although there had been plenty of fresh beef, there was scarcely a man who did not have a scorbutic taint. During the late civil war, scurvy, in some form or another, was generally prevalent in our armies. Though the statistics of the first two years show an extremely small number of cases, “unparalleled in the history of armies,” it is not a true exhibit of the actual prevalence of the disorder. There were reported in the first year, 1328 cases and 9 deaths, and in the second year, 7395 cases and 90 deaths: to which may probably be added 304 cases of purpura and 31 deaths (*Circular No. 6, Surgeon-General's Office, War Dept., 1865*). It first appeared to any extent in the Army of the Potomac, at Harrison's Landing, Va., in July, 1862, and from that time forward, until the end of the war in 1865, it continued an increasing and formidable disorder. “It occurred in all [the] armies subjected to hardships, especially in the West, and its worst and most fatal manifestation was made after the war had closed, and during the occupancy of the frontier of the Rio Grande by the twenty-fifth Army Corps,” and in consequence of the want of fresh vegetable food (*U. S. Sanitary Commission Memoirs*).

What has been written of scurvy in the British army, during the Crimean war, is equally applicable to our own, during the late war. “The returns convey but a faint conception of the disastrous part which it [scurvy] acted among the troops; for though it comparatively rarely presented itself in well-defined forms, and as an independent affection, yet the prevalence of scorbutic taint was widespread, and in a vast proportion of cases evident indications of it existed as a complication of other diseases, especially fevers and affections of the bowels” (*Medical and Surgical History of the British Army, 1858*).

In the first years of the war scurvy did not prevail amongst the Confederate troops apparently to any extent, for with a monthly mean strength of 160,231 officers and men, and with 1,056,349 cases of sickness and wounds entered upon the field reports in nineteen months, from January, 1862, to July, 1863, only 2203 cases of scurvy are recorded; and in 398,641 cases of sickness and wounds entered upon the hospital reports, there were only 2068 cases. But it progressively increased, with a diminishing commissariat and increased hardships (*J. Jones, in San. Com. Memoirs.*) The same authority, in his report on the Diseases, &c., of the Andersonville prison, states that scurvy, arising from sameness of food and imperfect nutrition, caused, either directly or indirectly, nine-tenths of the deaths amongst the United States army prisoners confined there.]

[**Morbid Anatomy and Pathogeny.**]—The days when scurvy was most prevalent were not those in which many post-mortem examinations were made, and our earliest knowledge of the morbid anatomy of

this disease is derived principally from Poupart and Lind. They tell us that in those cases in which flux or dysentery is absent, the intestines have been found perfectly sound, however copious the hemorrhage from them may have been. The principal effects of the disease were observed in all cases in the cellular tissue of the extremities. The quantity of congealed blood effused in that part, even where no stain or mark could be perceived on the skin, was quite astonishing. "It often lies," says Lind, "in large concrete masses on the periosteum, while the bellies of the muscles of the legs and thighs seemed quite stuffed with it, often an inch in thickness." He often found water effused into the cavities of the chest and abdomen, and no less frequently blood,—the quantity of blood effused in all parts sometimes amounting, in his opinion, to no less than a fourth part of that contained in the whole body. Poupart gives some further particulars, and says that on moving the limbs of some scorbutic patients a noise is heard; and that on examining the joints the epiphyses had entirely separated from the bones; and in other cases that the cartilages of the sternum had separated from their bones; and bones that had united after being broken, very often separated again at the site of fracture. He says that if we squeezed the ribs which had begun to be thus separated from their cartilages, "there came out abundance of corrupted matter, so that nothing was left of the rib but its bony plates." The mesenteric glands were usually enlarged; the spleen, often three times bigger than natural, fell to pieces, as if composed of coagulated blood. In two cases examined at St. Thomas's Hospital by the late Dr. Robert Williams, patches of ecchymoses were found under the pericardium covering the heart, and also under the arachnoid membranes covering the brain.

In some endemics of scurvy there is a marked tendency to the effusion of dark liquid blood into the serous cavities, and of the synovial membranes, the most common site being into the pericardium, then the pleura, and next the peritoneum (KARAWAGEN). The spleen is often enlarged, distended with blood, and soft. The texture of the muscular system is also soft and flabby.

[Haspel* and Marmy† give the following necroscopic phenomena of scurvy, most of which the writer has had the opportunity of verifying. Effusions, more or less blood-tinted, laminar, or in black grumous masses, into the subcutaneous intermuscular connective tissue, or, beneath the muscular aponeuroses. When occurring in the muscular substance, they compress, atrophy, or break down the primitive fasciculi. Occasionally they are subperiosteal, and then frequently cause local death of the bone. Their chief site is the hams, calves of legs, thighs, bend of elbow, pterygo-maxillary region, and groin. These effusions are fibrinous in character, more or less tinted with the coloring matter of the blood, and of variable consistence; they are either gelatinous, marked with pale yellow streaks, like heart-clots, or firm, elastic, yielding no fluid on pressure, and of a bright yellow color; from a quarter to one line in thick-

* [Gazette Médicale, 1859.]

† [Etudes Cliniques pour servir de l'Histoire du Scorbut et Typhus de l'Armée de l'Orient. Recueil de Méd. Chir., et Phar. Mil., 3e série, t. i, 1859.]

ness; and their capillaries capable of being injected, like other adventitious membranes—"scorbutic formation" (HEMMELSTIERN). There are serous or fibro-serous effusions beneath the arachnoid, or a reddish fluid, like currant-jelly water, is infiltrated into the subserous areolar tissue. Ecchymosed spots or patches upon the surface of the brain, and occasionally into its substance, particularly at the base, and near the pes hippocampi. Intra-pleural effusions. The lungs may be pale, bloodless, and gorged with serum; or there may be superficial violet stains or marblings on their surface; or wine-colored fluid in the pulmonary vesicles, intercellular passages, and subserous connective tissue. In those cases where special lung symptoms have been present, portions of the pulmonary substance, generally in the lower lobes, are gorged with black blood, and are inelastic, uncrepitant, and impermeable. Again, there may be ecchymosed spots in the interlobular connective tissue; or large, fluctuating grumous masses, in defined, irregular cavities, not lined, may exist in the lung parenchyma. Occasionally there is gangrene of the lung. Ecchymosed spots or patches in the subserous pericardial tissue, on the surface of the heart, and intermuscular connective tissue. Spleen softened. On the mucous and in the submucous tissues of the stomach and bowels, ecchymoses, with often ulcers of the large intestines. (See *Chronic Camp Dysentery*, p. 590, vol. i.)]

The blood appears to be deficient in red particles (BUSK, BEQUEREL, RODIER, ANDRAL, FRICKS), and does not impart a stain to the lining membrane of the heart and great vessels. A fluid or dissolved condition of the blood has also been noticed, appearing often as "a mere gore, not separating into crassamentum and serum, and putrefying soon. It appears to be starved of some essential ingredient." It flows with difficulty from the vein, and, after standing some hours, deposits a thick, muddy sediment, which subsides from a reddish serum; and in the last stage of the disease it becomes quite black (ROUPPE). There does not, however, appear to be any evidence of deficient powers of coagulation in scorbutic blood; and so long ago as 1699, Poupert noted the large coagula found after death in the cavities of the heart. Three analyses of the blood were also made by Mr. Busk, before 1840, in all of which the quantity of fibrine was found to be above its normal standard. Stoeber, in 1845, came to the same conclusion; and so did Andral, on repeating analyses which at first led him to an opposite conclusion. Two facts of great importance, if confirmed, have been observed by Chatin and Bouvier. They have observed that the *albumen* of the blood does not coagulate under a temperature of 74° Cent., or 165.2° Fahr.—that is, from 5° to 8° Fahr. above the normal standard; so that the albumen of the blood undergoes some change in scurvy which increases its solubility. They also observed that the force of cohesion of the *fibrine* was so much lessened that they were unable to isolate it thoroughly from the red corpuscles, and it was this attraction of the fibrine and the red corpuscles which the early writers on scurvy attempted to describe by the terms "*agglutinated blood*," "*viscid and thickened crassamentum*" (Parkes "*On the Pathology and Treatment of Scurvy*," *Brit. and For. Med.-Chir. Review*, Oct., 1848). Thus all the phenomena of scurvy, and the conditions under which it becomes developed,

point to the blood as essentially altered. What the alteration definitely consists in has not been yet determined; but it appears that the condition of the blood in scorbutus does not consist in the want of a due proportion of either of its three prominent constituents. The proportion may in some cases be deranged, but it appears that in all cases there is a deficiency or altered quality of some essential ingredient. The following statements by Drs. Copland, Watson, and Wood, contain a summary of the opinions of the chemical pathologists of the present day who have specially investigated the nature of scurvy by analysis of the blood:

Dr. Christison supposes that scurvy arises from the want of vegetable albumen or animal casein in the food; that it is the deficiency in the quantity of azotized aliment, and consequent insufficient nourishment of the body, which is the cause of scurvy. Dr. Garrod believes that the malady is caused by the absence of *potash*, and that potatoes and other antiscorbutics owe their virtues to the *potash* they contain. The following are his conclusions: (1.) That in all scorbutic diets *potash* exists in much smaller quantities than in those which are capable of maintaining health; (2.) That all substances proved to act as antiscorbutics contain a large amount of *potash*; (3.) That in scurvy the blood is deficient in *potash*, and the amount of that substance thrown out by the kidneys is less than that which occurs in health; (4.) That scorbutic patients will recover when *potash* is added to their food, the other constituents remaining as before, both in quantity and quality, and without the use of succulent vegetables or milk; (5.) That the theory which ascribes the cause of scurvy to a deficiency of *potash* in the food is capable of rationally explaining many symptoms of that disease. These propositions of Dr. Garrod's require much further investigation, and they are not supported by the most elementary and important facts in the history of scurvy.* Both soda and *potash* are constant constituents of the animal body, and it appears that they are not capable of replacing each other. For example, we always find the *potash* to exist in large quantities in the ash of muscle, soda in very small quantities (BERZELIUS, LIEBIG). In the ash of the blood we find the relation reversed. It appears, also, that the muscular system requires the presence of *potash*; and we should therefore expect to find that where there is a deficient supply of this base, the effect would soon be manifested in the functions of that system. This we do not find to be the case in scurvy. Without any amount of wasting of the body, we may find marked muscular debility; but men in an advanced stage of *scorbutus* have been known to do severe labor, till even the approach of fatal syncope from the weakened action of the heart; and this, perhaps, is one

* ["Brought forward by Garrod as an hypothesis, and based, not on analysis of scorbutic blood and tissues, but on the fact that the food which produces scurvy contains less *potash* than the well-known antiscorbutic foods, this hypothesis has been accepted far too readily as the true cause of scurvy. It has not been shown yet by analysis that the food of a scorbutic patient has been actually deficient in *potash*, nor have the analyses of the urine or sweat proved that the natural elimination of the *potash* has been at all altered. Therapeutic trials with the salts of *potash* prove that *potash per se* has no antiscorbutic power." (*Pract. Hygiene*, 2d ed., p. 464.)]

of the earliest symptoms of the disease. "Opposed also to this theory," writes Dr. Wood, "are the facts that nitrate of potash has often failed to cure the disease, while it has been cured by the addition of pure citric acid; and that, after failure under treatment with the salts of potash, recovery immediately commenced when the patient was allowed to eat fresh vegetables." Trotter also always held the opinion that the real antiscorbutic principles in fruit and vegetables were the vegetable acids, particularly citric, oxalic, and malic acids; and to test the accuracy of his opinion, he made, in 1800, a series of comparative trials between *lemon-juice* and pure *citric acid*. Both these remedies were furnished to eight or ten ships, and reports were then obtained of their effects. The result was that *citric acid* was found the most efficacious. Sir William Burnett, also, was in the habit of supplying convict ships with *citric acid* and *nitrate of potash*, as well as with *lemon-juice*, in order that comparative trials might be made of the relative value of these remedies; and the official documents bearing on this point were carefully examined by Dr. Parkes, who considered that nothing could be more convincing than the evidence they contained, showing that the efficacy of *citric acid* was clearly proved, while *nitrate of potash* was shown to be inferior in curative power (*On the Pathology and Treatment of Scurvy*, l. c.).* The astonishing effects of fresh *lemon-juice* have been evidently underrated by Dr. Garrod; and it is more probable that its virtues are due to citric and other acids than to potash. Dr. Aldridge contends for the influence which should be ascribed to a deficiency of phosphorus, sulphur, lime, and the alkalies, in occasioning scurvy. That something may be owing—a part merely—to the causes contended for by Dr. Aldridge is not improbable. But it is unnecessary to pursue this subject any further than very briefly to state, that one of the most evident changes from the healthy condition is seated in the blood, which is altered in composition either by the addition to it of some ingredient or ingredients, or by the absence of something which ought to exist in it; and the deficient ingredient may be one of the ordinary constituents of the blood, or it may be some principle or element entering into their composition. This deficiency is due to the absence of certain articles of diet; and the disease is known by experience to be at once cured by supplying those articles.

Symptoms.—The earliest are a change of color of the skin, particularly of the face and eyelids.

[Sometimes the condition of the eye and its appendages is the first and only physical sign of the disorder. The skin around one or both orbits is puffed up into a bruise-colored swelling; the sclerotic conjunctiva is tumid and of a brilliant red color throughout, and elevated about one-eighth of an inch, the cornea appearing at the bottom of a circular well. There is no pain or discharge. It betokens a serious form (BIRD, BELLINGHAM). Breathlessness is often one of the initial symptoms.]

* [Nitrate of potash is inefficacious against scurvy (MURRAY, BRYSON). The Ottawa lumberers living on pork salted with nitre suffer greatly. Dr. J. O. Grant found in one shanty out of 86 of these men 25 affected with scurvy. (*Med. Times and Gaz.*, vol. ii, 1868.)—EDITOR.]

At the same time there are vague, wandering, rheumatic-like pains in the limbs, weariness, depression of spirits, and a longing for fresh vegetables and fruits, [about which there are often lively dreams (KANE).] With these pains, however, there is no fever; the pulse is soft and natural, and the temperature of the body lower than usual, and great disorder of the respiratory function may supervene. The countenance appears pale or yellow, and bloated [and dejected]; there is great depression of the physical powers, followed by swelling of the gums, which become soft, spongy, and hang over the teeth in large fleshy-like palmated masses, very much disposed and readily excited to bleed; and the edges of the gums are purple where they are in contact with the teeth [or white-mottled]. Several old observers state, and Fauvel confirms the observation, that in old people without teeth these vegetations do not occur, and the gums remain comparatively unaffected. In one case he noticed that a single remaining tooth was surrounded by a mass of swollen gum; the tooth was extracted, and the gum immediately became level and firmer, while the other symptoms preserved their intensity. The swollen and fungous gums, as Dr. Parkes observes, have always appeared to be much more intense in cases occurring on shipboard; and it is not improbable that they are affected peculiarly because they are exposed to pressure and attrition.

[Ulceration of the gums, at their free border, may occur. Sometimes, while most of the characteristic phenomena are present, the gums are unaltered, except being paler than ordinarily (SHAPTER, BELLINGHAM, PERRIN, BUZZARD).]

All parts suffer from pressure in cases of scurvy,—the merest rub causes an ecchymosis, the slightest possible blow produces an extensive bruise, and the gums may suffer most from the causes already noticed. A small eruption (like flea-bites) of a purple hue is next seen on the lower extremities; and about the same time the muscles of the leg or thigh become hard and painful, and in a day or two the skin over the pained part becomes first yellow and then purple. This discoloration forms patches sometimes as big as the palm of the hand, and may extend over half the leg and thigh. The popliteal regions are a frequent site of this pain and discoloration, sometimes attended with œdema, especially of the ankles and feet. The discoloration is especially apt to show itself in the situation of old cicatrices, and injuries.

[These extravasations are both *superficial and deep-seated*. The *superficial* are, (1) *dermic*, and (2) *subdermic*. The *dermic ecchymomata* vary in size from small, dark-colored spots to large blotches; they may be distinct, or may come together at some point of their circumference. The anatomical site of the spots is the hair follicles or sudoriferous canals, and their structure hinders the diffusion of the sanguine serosity. The epidermis becomes dry, rough, and raised in scales, resembling the skin of a recently-plucked fowl (H. LARREY), and the sudatory function is diminished or suspended. Subsequently desquamation takes place. The solid part of the blood may, however, remain infiltrated in the meshes of

the derm, forming brown spots, which may last for months, the epidermis over these being smooth and shining.

In the *subdermic ecchymomata* the blotches are larger and of variable depth. They may invade the whole limb, but the most common site is the ham, groin above and below Poupart's ligament, the thigh, calf, and pterygo-maxillary region. If they compress superficial veins, they cause œdema and pain. They produce swellings in the flexures of the joints, most often in the ham, or shin, in the pterygo-maxillary region, and bend of the elbow. They are hard, but pit on persistent and firm pressure. When on the shin and circumscribed, they may be mistaken for syphilitic nodes. Resolution is their usual termination, their hue changing from dark purple to green and yellow. When the dark color persists with a greenish-yellow border, it is evidence that the effusion is very thick. Should their termination be by ulceration, a sort of indolent boil forms, with a deep-red base and black summit, with œdema of the adjacent connective tissue. If the surface is galled, there is constant weeping of a sero-sanguinolent fluid; the tumor flattens a little; and a small, dark, ulcerated central point, with wine-red edges, appears: this is a gangrenous mass, which gains little by little, until an ulcer of variable size is established. The most frequent site of these scorbutic ulcers are the calves of the legs, the buttocks, thighs, sacrum, shoulders, arms, and more rarely the chest and abdomen. The *deep-seated extravasations* are, (1) *sub-aponeurotic*; (2) *parenchymal*, into the muscles, kidneys, heart, lungs, &c. These extravasations into the muscle-tissue never end in suppuration, but the tissue becomes atrophied, and undergoes a sort of gelatinous degeneration; resolution is very sluggish. When the effusion is beneath a resisting aponeurosis, as the fascia lata, there is no alteration in the integument immediately over it, but the swelling and discoloration happen at remote points, as the ham, about the knee, &c. The source of these extravasations would seem to be the softened capillary vessels, permitting the leakage of altered blood; there is no apparent solution of continuity except an ulcer exists (MARMY, *loc. cit.*.)]

The tongue is now white, the breath fetid, and the stools generally pale.

[Ptyalism may take place, with swelling of the parotids and submaxillary glands, and soft, swollen, or ulcerated gums and tongue, giving the look of mercurialization (RITCHIE).]

As the disease advances, all these symptoms are aggravated. The loss of physical power increases, the purple spots have a tendency to ulcerate, and the ulcers are distinguished from all others by their putrid fungoid appearance and great tendency to bleed; old sores open, and the callus of broken bones has even been dissolved and their ends separated.

[The mucous membrane of the gums, cheeks, and even pharynx, sphacelates in shreds. The cheek may be destroyed in spots, with constant leakage of a fluid composed of saliva, blood, and gangrenous tissue, exhaling a putrid odor. In such cases a diphtheritic angina is apt to intercur, with œdema of the glottis, and is usually fatal.]

Profuse hemorrhages frequently take place from the mouth, nose, [pharynx], lungs, [genito-urinary organs], or bowels.

[The extravasated fluid raises a bulla on the mucous membranes, which grows larger, and, finally, breaking, blood is poured out (MARMY).]

The teeth become loose, so that they either fall out or may be taken out by the finger and thumb. The pulse hurries on to 120 or 140, and at length the patient sinks from diarrhœa or dropsy, and with effusion so sudden that he perhaps has walked a short distance and then died in a quarter of an hour afterwards.

[Albuminuria is not infrequent, and is due to congestion of the kidneys, as seen in the autopsies, with extravasations, sometimes, into the renal tissue.]

The *duration* of the disease is generally many weeks, and sometimes under the most favorable circumstances many months, the patient recovering his strength very slowly.

When the disease follows ague, obscure or irregular remissions or intermissions of febrile symptoms are observable, and more or less enlargement, with pains in the region of the spleen, is often detected. From the commencement of the disease the alvine evacuations are more or less disordered, but the change may not attract attention. At first costiveness prevails, but subsequently the stools become much more frequent and very offensive. Diarrhœa, with colicky pains, supervenes, and, with more or less attendant hemorrhage, rapidly sinks the patient beyond hope of remedy. Dysentery frequently supervenes upon the scorbutic state, more especially when dysentery is epidemic, and this combination of maladies is sometimes termed "*scorbutic dysentery*." The tongue, pale at first, becomes broad, flabby, and indented at the edges of the teeth. The great poverty of the blood causes the mucous membrane of the tongue and inside of the lips and cheek, as well as of the skin, to have a peculiar paleness, which contrasts remarkably with the appearance of the gums. The eyesight is frequently weakened, and night blindness (hemeralopia or nyctalopia) has frequently been observed as a commencing and concomitant symptom [—the ophthalmoscope showing, however, no morbid changes of the deep tissues]. A most distressing state is the inability to sleep at night.

"The tendency to *swoon* in the most severe cases is sometimes so great that the slightest motion, or the erect posture, or even any trifling exertion, may be followed by fatal syncope. It is stated in the account of Lord Anson's voyage, that many of the men, although confined to their hammocks, ate and drank heartily, were cheerful, and talked with much seeming vigor and in a strong tone of voice, and yet, on their being the least moved, although it was only from one part of the ship to the other, and that in their hammocks, they have immediately expired; and others, who have confided in their seeming strength, and have resolved to get out of their hammocks, have died before they could reach the deck. And it was no uncommon thing for those who could do some kind of duty, and walk the deck, to drop down dead in an instant, on any endeavor to act with their utmost vigor" (COPLAND).

[There is an intercurrent chest affection often happening in scurvy, and which may be mistaken for pneumonia. It occurs during cold and

damp spells. It begins with slight rigors, followed by feverishness, and accompanied by lancinating pains in one or both sides. There is dyspnoea, and a feeling of constriction in the chest, as if a cord were drawn tightly round it. This condition is commonly caused by intra-pleural effusion of blood, but sometimes blood escapes into the pulmonary tissue; in the latter case, the expectoration becomes dark and sanious, and has a fetor resembling that of pulmonary gangrene, but which is due to decomposition of the sanguine fluid. Cold sweats, increasing dyspnoea, anxiety, and a frequent, thready pulse, precede death, the constant termination. Sometimes there is neither pain nor cough, but rapid increase of the breathlessness, and sudden death. The physical signs of the lung trouble are often wanting, though sometimes there may be dulness, mucous rhonchi, and bronchial respiration (HASPEL). Chest dulness on percussion in scurvy, may sometimes be due to sanguineous effusions into the thoracic muscles (HASPEL, BUZZARD).]

With our knowledge now of the phenomena of embolism, may not the suddenly fatal end of such cases be due to the morbid condition of the fibrine of the blood already referred to?

Diagnosis.—The scorbutic state of the skin is to be distinguished from *flea-bites*, *bruise*, *typhus fever*, and from *purpura hæmorrhagica*.

[A careful examination of the skin and gums, with a history of the case, will prevent its being mistaken for *chloro-anæmia*, though the color of the skin in the latter disorder, particularly when dirty, resembles that of a scurvy patient, but the blotches are absent. In *purpura* the blotches, though closely resembling those of scurvy, occur suddenly, in persons previously in apparent good health; while in scurvy they have been preceded by pallor and listlessness. In *purpura*, the dingy hue of the skin is wanting, also the articular effusions, and pains in the limbs.]

Prognosis.—In the present day, when the patient can command medical care and proper diet, scorbutus, though tedious, is seldom fatal. When these, however, have been wanting, the mortality has been terrible. Lord Anson, it should be remembered, in his voyage round the world, lost above 200 men, and at last could not muster more than six fore-mast men in a watch fit for duty. At the commencement of our last war with France, on the fleet returning from sea, it often happened so many men were landed ill of scurvy that even Haslar Hospital, large as it is, could not contain them, and many were lodged in the chapel, others in tents, while others died in the boats before reaching the shore.

Cause and Conditions under which Scorbutus is Developed.—In the Middle Ages scurvy prevailed to so great an extent that it was said to be epidemic among the inhabitants of the low countries of Holland, Friesland, Brabant, Pomerania, Lower Saxony, and, indeed, all countries from the 50° to the 60° of north latitude. This has been attributed to the absolute want of winter food for the cattle, so that it was necessary to kill them on the setting in of the frost, and either to salt or dry the flesh. Food was deficient and of improper quality, and hence the large stores of salt provisions found in the larder of the elder Spencer in the days of Edward II, even so late in the spring as the 3d of May. Six hundred bacons, eighty

carcasses of beef, and six hundred of sheep, was his abundant supply. In all these countries, however, in proportion as agriculture has advanced, and a succession of green crops has enabled the farmer to kill his best and fattest meats in winter, and in proportion as fresh vegetables have been introduced at our tables, together with a liberal use of wine and beer, so has this disease disappeared. The former universal prevalence of scurvy in the Navy, and its almost entire disappearance in the present day, necessarily has reference to a particular cause—the too exclusive use of salt provisions and absence of fresh vegetables. “In 1797 the victualling of the Navy was changed, greatly improved, and strictly regulated; and immediately consequent to the change the health of the seamen improved strikingly. Scurvy, typhoid fever, dysentery, and putrid ulcer, which up to the period of the change produced great havoc, became comparatively rare in occurrence and light in impression.” Since 1797 the improvements have consisted in giving cocoa instead of gruel for breakfast, issuing salt meats at a much earlier period after being cured, the supply of better articles, and in greater abundance by one-third, the substitution of tea in the afternoon instead of spirits; but, chief of all, the use of fresh vegetables as often as possible: and, with every improvement in these respects, there has been, as a general result, a further improvement in health, till these four forms of disease, at no distant date so destructive, are scarcely known except by name.

It is now, however, completely established that salted meats are not more productive of scurvy than fresh meats, so far as concerns a monotony of diet. The experience of the Russians in 1720 and 1736, of the French in 1750–60, and of our own regiments at the Cape in 1836, sufficiently establishes this point.

[There is no doubt, however, that in the great majority of scurvy outbreaks, salt meat has formed an important part of the food taken by the sufferers.

Liebig has shown that the process of salting meat deprives flesh of a large proportion of its most important constituents, so that the remainder is deficient in nutritive properties; and the altered and hardened character it acquires, renders even such nourishment as it contains difficult of assimilation.]

Besides the injurious effects of cold and moisture, as well as impure air, combined with the conditions already noticed as tending to favor the development of scurvy, it is now well known that exposure for a lengthened period to the pernicious influences of a malarious district greatly aids in developing scorbutus. By observations especially devoted to this subject, I have determined that, amongst our troops who had been in Bulgaria during the war with Russia in 1854, there were two and a half per cent. of admissions for scurvy amongst them more than among those who served in the Crimea only; and that the deaths among them reported from this disease were also greater by three per cent. than among those troops who served in the Crimea (*Glasgow Med.*

Journal, July, 1857; and *Trans. of the Royal Med.-Chir. Society*, vol. xl.)

Recent combined researches have shown that scorbutic diseases were developed amongst our troops in the Crimea under the following conditions: (1.) Deficiency of absolute nutriment; (2.) Improper adjudication of the *nutrient* and *respiratory* principles of the *diet*—its monotony; (3.) Bad quality of the diet, and improper cooking, or none at all; (4.) Exposure to cold, combined with imperfect clothing, and labor beyond the strength of the best fed men; (5.) The persistent pernicious influence of residence in a paludal district (Bulgaria). But these circumstances are only to be regarded as the *occasional antecedents*, any one of which, or all combined, can never, *per se*, originate the disease. LOOKING TO THE HISTORY OF SCURVY, IT WILL BE SEEN THAT THE INVARIABLE AND INDISPENSABLE ANTECEDENT OF THAT DISEASE HAS BEEN A DEFICIENCY OR ABSOLUTE WANT OF FRESH VEGETABLE FOOD. Privation of vegetable food is its one essential cause. There is no other invariable antecedent; and there are sufficient reasons why it may not always be followed by scurvy. It is this antecedent which is the *vera causa* of *scorbutus*; and the most successful methods of prevention and of cure are in accordance with the hypothesis which assumes it to be the cause (BUDD, CURRAN, LAYCOCK, PARKES). "The giving of vegetable food is its one essential counteractive" (SIMON).

[In 1846, the potato crop failed in Great Britain and Ireland; and in the following year there was much scurvy amongst all classes. There was an outbreak of the disorder in Scotland, especially amongst the artisans and the laborers on the railways, in 1846–47, described by Drs. Christison, Ritchie, and Lonsdale (*Ed. Monthly Journ.*, 1847), and the general fact with regard to the food of all was, that it failed in variety, and in the quantity of its animal constituents; none had tasted potatoes after the harvest of 1846—a period of seven or more months—nor fresh vegetables; but animal food, fresh and salted, was taken in large quantities, as well as pea-soup, suet puddings, bread, and oatmeal. At Workington, a seaport town of 7000 inhabitants, there was no case of scurvy, turnips having been used in large quantities. Dr. Curran (*Dublin Quar. Jour.*, 1847), describing the disorder as it appeared in Ireland, says: "In no single instance could I discover that green vegetables or potatoes had formed any part of the regular dietary;" grains, tea or coffee, flesh and fish, being the food. Dr. Shapter observes, that in Exeter "the only difference in the usual diet of the sufferers consisted in the absence of the potato," and that many of them had abundance of the necessaries of life, except fresh vegetables (*Med. Gaz.*, vol. iv).

In the Crimean war, the allied armies suffered severely from scurvy. In the British army it first appeared in Bulgaria, where the diet was poor, and the supply of vegetables scanty. When it arrived in the Crimea, there was an abundance of grapes, cabbages, &c., and though the ration was inferior, the disorder disappeared; but as winter set in, and vegetable food could no longer be got, it began again. When the supply of fresh vegetables and lime-juice became more constant, it gradually disappeared, and there were but few cases during the second winter.

The French army suffered still more, no less than 23,000 cases of scurvy being recorded (SCRIVE). Good but lean fresh meat was issued, at first

twice, then five times a week, with rice, and occasionally dried vegetables—chiefly peas, beans, and lentils—in small proportions, and bread irregularly. In the spring, with vegetation, the number of cases decreased; but in July, with a parching sun, the disease reappeared, and in the course of three months, the finest and warmest in the year in that region, no less than 5000 cases of scurvy occurred (BUZZARD). The Sardinian army, on its arrival in the early part of the summer of 1855, was largely affected with scurvy, which was checked by the issue of fresh vegetables. Dr. Buzzard, who was a staff-surgeon in the army of Omar Pasha, says, that "loss amongst the Turks from this disorder was still greater, and that the original force which formed part of the expedition from Bulgaria to the Crimea, was almost entirely swept off by disease, of which scurvy formed an important element." (Reynold's *System of Medicine*, Art. Scurvy, vol. i, 1866.) During the winter of 1854-55, of Omar Pasha's troops as many as 1000 were sent away monthly, all suffering severely from this disorder. During the summer of 1855, this army was encamped near Balaklava, and Dr. Buzzard remarks, that though "their food was very imperfect, they were supplied with onions, and consumed large quantities of watermelons," and no cases of scurvy appeared, nor could he detect any scorbutic taint in patients suffering from other diseases. But during the winter all vegetable supplies ceased, and "shiploads of sick were brought to Trebizond, all of whom were severely afflicted with this disease;" in some cases "developed to an extent which recalled the terrible descriptions of the disorder contained in the narratives of our early voyagers." This was explained by the fact, that besides the absence during the winter of fresh vegetables, they had not had a sufficient quantity of food, their diet consisting "entirely of biscuit, a little rice, haricot beans, and 'yagh' (a coarse butter made from mutton fat)," and there was "absolute starvation." In 1836, over 100 cases of scurvy occurred in the 75th regiment while quartered at Caffreland, when not an ounce of salt provisions was issued, when the men had no harassing duties, and were abundantly supplied with fresh meat. The Hottentot troops doing duty with them were served with the same rations, but sought out for themselves pumpkins, melons, wild fruits, and esculents, and entirely escaped; as did, also, the 27th and 72d regiments, encamped eighteen miles distant, fed with the same rations, but supplied with vegetables in addition. Dr. Buzzard states that scurvy is common in North Wales, where fresh meat and milk are abundant, but where there is little or no garden produce, and that he has met with many cases amongst the poor of London, who have eaten no meat of any kind for weeks, but had lived on tea, bread, and butter. At the close of the Punjaub campaign (1848-49), the troops had abundance of excellent fresh meat and bread, but no fresh vegetables, yet suffered severely from scurvy; and in the Himalayan stations, during the second Burmese war, under the same circumstances, scurvy was prevalent amongst the troops (*Med. and Surg. Hist. of the British Army*, vol. ii, 1858).

Scurvy, as we have seen (*ante*, p. 747), prevailed to a great extent in the United States Army during the civil war. This was not owing to any deficiency in the ration, or to the want of liberal provisions for fresh vegetables by the government and private organizations, or the neglect of the medical department in issuing antiscorbutics. All these were most generously provided. But there were times when they could not be obtained, or used; when the men were obliged to live on marching rations; or, when, through the ignorance or negligence of commanding officers, they were not distributed. Scurvy, under these conditions, was the invariable

sequence. At one time, in the Army of the Potomac, when "symptoms of scurvy began to appear," and there was a general "low vitality of the men," Dr. Letterman found that he had rightly attributed it to "want of fresh vegetables;" for, "while large supplies of potatoes had been issued, the troops received in some cases a very small quantity, and in others none at all" (*loc. cit.*, p. 106). Again he says (pp. 109-10), "This favorable state of the health of the army, and the decrease in the severity of the cases of disease, are, in a great measure, to be attributed to the improvement in the diet of the men, by the issue of fresh bread and fresh vegetables, which has caused the disappearance of the symptoms of scurvy, that in January began to assume a serious aspect." The testimony of Dr. F. Hamilton, with respect to the Army of the West, is to the same effect (*loc. cit.*.)]

Treatment.—The early history of navigation, as it records the greatest ravages of scurvy, so does it also record the best antidote to the disease. Of four ships which sailed from England in the beginning of April, 1609, for the establishment of the East India Company, they were all so severely visited by scurvy as to have lost nearly one-fourth of their crews when they arrived at the Cape of Good Hope. The crew of the Commodore's ship was not attacked. This immunity arose from three tablespoonfuls of lemon-juice having been served daily to each of his men. But notwithstanding this evidence of the success of lemon-juice in preventing scurvy—evidence the most conclusive—this valuable remedy and preventive was altogether slighted for a hundred and fifty years afterwards (COPLAND). Lord Anson's people, in 1740, on reaching the Island of Tinian, were recovered principally by eating oranges; and that noble, brave, and experienced commander was so convinced of their usefulness that, before he left the island, he ordered one man from each mess to lay in a stock for future security. Sir Charles Wager's people, also, were terribly afflicted with scurvy in the Baltic. Sailing, however, in the Mediterranean, and having heard how effectual oranges and lemons were in the cure of this disease, he took on board at Leghorn a large quantity of them, ordered a chest each day to be brought on deck, and allowed the men, besides eating what they chose, to mix the juice with their beer, and to pelt each other with the rind, so that the deck was strewn with the fragrant liquor. By these means he brought his men home in good health.

In the year 1747 Dr. Lind made some comparative trials between this and some other modes of treatment (as vinegar, sulphuric acid, and tamarinds) on board the "Salisbury," at sea. As a general conclusion from his experiments, he affirms that orange- and lemon-juice, or more properly the citric acid obtained from all the species of the botanical genus *citrus*, or the natural order of fruits called *hesperidæ*, are greatly more efficient than any other remedy in the cure of scurvy.

Notwithstanding this strong opinion of Dr. Lind, the Navy continued to suffer severely from scurvy for half a century, till the Admiralty gave a general order for the supply of lemon-juice. This salutary measure was accomplished by a representation from the

Medical Board of the Navy, in the year 1795, when Lord Spencer was First Lord of the Admiralty, after a trial made on board the "Suffolk," of seventy-four guns. This ship sailed from England on the 2d of April, 1794, supplied with a quantity of lemon-juice sufficient to serve out two-thirds of a liquid ounce daily to every man on board, and this was mixed with their grog, with two ounces of sugar. She arrived at the Madras roads on the 11th of September, after a passage of twenty-three weeks and one day, without having had any communication with the land, without losing a man, and having only fifteen on the sick-list. Scurvy appeared in a few of the men during the voyage, but disappeared on an increased dose of lemon-juice being administered. "Let this fact," says Sir Gilbert Blane, "be contrasted with the state of the Channel fleet in 1780, when Admiral Geary's fleet returned into port, after a ten weeks' cruise in the Bay of Biscay, with 2400 men ill of scurvy; and let the state of this fleet be contrasted with that of the Channel fleet in 1800, which, by being duly supplied with lemon-juice, kept the sea four months without fresh provisions, and without being affected with scurvy."

In 1780 the number of cases of scurvy received into Haslar Hospital was 1457; in 1806 *one* only; and in 1807 also *one*. While it is notorious that many medical men have never seen the disease, it is, as Dr. Budd has assured Dr. Watson, by no means rare in the hospital ship at Greenwich, which is often full of cases of scurvy; most of the patients so affected having just arrived in *merchant ships* from a long voyage; and some rumors are now abroad to the effect that scurvy is more frequent in the merchant service than heretofore—and, if so, the least that can be said is, that it is a disgrace to the merchant princes of a nation like Great Britain.* The

* With regard to the prevalence of scurvy among sailors in the merchant service, Dr. Barnes (Physician to the "Dreadnought" hospital ship) reports in 1864 to Mr. Simon that "of the entire number of cases admitted during the last twelve years into the Sailors' Home at Poplar, nearly half are, at the time of their admission, suffering more or less from scurvy, and of these perhaps a twentieth part seriously diseased with it; that of the entire number of cases admitted during the last twelve years into the hospital ship 'Dreadnought,' cases of scurvy have formed a twenty-fourth part; that to these must, of course, be added sufferers who are not taken into such establishments—a large but uncertain number of cases taken into the low lodging-houses of the water side; but that this is for London alone, and is not nearly all due to London shipowners; that of eighty-six cases of scurvy treated in the 'Dreadnought' during 1863, only fourteen came from ships which had issued from the port of London, while thirty-one came from foreign ships, twenty-one from Liverpool ships, eight from Sunderland ships, and twelve from Glasgow and other British ships; that Liverpool ships, besides furnishing to London a large proportion of the scurvy which is treated here, convey probably a much larger quantity of the disease to their own port of departure; that at all events, during 1863, fifty cases (all probably severe) were admitted at the Liverpool hospitals, and during 1862 a dozen cases at the Glasgow and Greenock hospitals; that shipowners of Liverpool and other northern ports, and of Hamburg and America, are those who exhibit the greatest amount of disregard of the safety and health of their crews. The plight in which the poor sailors from certain services are admitted is pitiable to witness. Disabled by hardship, semi-starvation, and ill-usage of every kind, they are cast out with the same indifference with which a worn-out block would be thrown overboard." When it is remembered that the security of this country has on several occasions been imperilled by the disablement of the Royal Navy through scurvy, it may be presumed

disease, "so fatal when left to itself, is cured with the greatest facility. Symptoms apparently the most grave and serious vanish as if by magic, and without leaving behind them any serious injury to the constitution. The sanious discharge from scorbutic sores has been known to change color and to become healthy in a few hours after the commencement of treatment. In pure cases of scurvy the blood, and the blood only, is at fault." (PARKES, *l. c.*) "Lemon-juice," writes Dr. Watson, "is really a specific against scurvy, whether it be employed as a preventive or as a remedy.* It supplies something to the blood which is essential to its healthy properties." The potato seems to be no less efficacious as a remedy and preventive (BUDD). The reader will find a most interesting account of the efficacy of potatoes and of onions in Dana's *Two Years before the Mast*—a book well worth reading. The antiscorbutic principle, whatever it may be, is in greatest amount in unripe fruits; it lessens gradually as they ripen; and if the juice be obtained, it disappears when fermentation occurs. When lime-juice becomes musty, a mucilaginous principle is developed at the expense of the citric and malic acids; and the *percentage* of citric acid gradually decreases (SIR WILLIAM BURNETT, PARKES). Good *lemon-juice* seems to be more effectual, however, than pure *citric acid*; probably from its containing *malic* and *tartaric acids*, besides *citric*, and from the *citric acid* being in the form most easily absorbed and decomposed by the digestive organs of man. The *Materia Medica* gives numerous analogous examples of the superior efficacy of a medicine in its *natural combinations* (PARKES). This is all we can yet say, however, regarding the actions of either of these means of cure, notwithstanding the researches of the chemists of the present day. Moreover, it is sufficient: and with such remedies at command, the prevalency of scurvy in *merchant vessels*, or in any navy, ought not to exist. "The one thing wanted in order that scurvy should be entirely banished from the *mercantile marine* is proper provision for the dietary of the crew, —such provision as is enforced in *emigrant ships*, where each person's weekly allowance must have in it 8 oz. of preserved potatoes, and

that the same cause will imperil the safety of our merchant ships. And there can be no doubt that many ships have actually foundered at sea because the crews were so prostrate from scurvy as to be unable to handle them when overtaken by severe weather. It has been the custom to inquire what proportion of the crews were disabled from scurvy. As might be supposed in the case of a disease resulting from a cause operating upon the entire crew, this proportion is often very large. Thus there were recently admitted on board the "Dreadnought" twelve cases of severe scurvy from one ship; two others were known to be seriously affected; and the entire complement of officers and men was only nineteen, leaving but five men in all able for duty to work the ship. The proportion of crew disabled has ranged from 20 per cent. to 70 per cent.; and it is certain that scurvy ships have rarely a hand to spare. Deprive such ships of a fourth, a half, or two-thirds of their force, and the peril of a ship, cargo, crew, and passengers, in stress of weather, is obvious; and it must not be forgotten that where scurvy has prostrated a large part of the crew, the vigor of the remainder is sure to be sapped, so that there may not remain a single sailor before the mast in a state of thorough efficiency (Simon, in *Sixth Report on Public Health*, p. 19).

* [It is essential that the lemon-juice should be *pure*; and to insure this it should be analyzed, before being received either in the army or navy. Ten per cent. of brandy (s. gr. 980) or rum (s. gr. 899) ought to be added; and it should then be packed in jars, covered with a layer of oil, and sealed.—EDITOR.]

3 oz. of other preserved vegetables (carrots, onions, turnips, celery, and mint), besides pickles and 3 oz. of lime-juice; where, also, there is considerable variety of breadstuff; and where, on two days in the week, preserved (not salted) meats must be given. With such a dietary as this, the details of which might be varied, provided its principle were adhered to, the occurrence of scurvy would be impossible. And even with dietaries inferior to that just described, scurvy would not occur as it does, if but the provisions of the 'Merchant Shipping Act' (17 and 18 Vict. c. 104) were obeyed,—that whenever a crew shall have been consuming salt provisions for ten days, lime-juice or lemon-juice and sugar shall be served out at the rate of half an ounce each per day; and if, during the voyage, the opportunities which offer themselves were fairly used for getting new supplies of fresh animal and vegetable food; but owners disobey the law, and captains neglect opportunities to cure the results of this disobedience. Owners, notwithstanding the law, will send forth their ships on long voyages without any provision of lime-juice, or with lime-juice insufficient in quantity, or with lime-juice of which the quality is bad; and captains, with half their crews more or less disabled, are known to run past St. Helena or the Western Isles, when a few hours' delay would obtain sufficient provisions to repair the mischief occasioned by first neglect" (Simon and Barnes's *Sixth Report on Public Health*, 1864, p. 20).

It is perhaps hardly fair to attribute the improved health of the Navy entirely to the introduction of a daily allowance of lemon-juice, considering that the quantity of the diet was greatly increased, and its quality greatly improved, contemporaneously with this addition. It is gratifying, however, to see how largely these combined measures have improved the health of the Navy, and rewarded the cares of those who superintend it; for during the nine years preceding these changes the sick seamen sent to the hospitals were 1 in 3.9, while in the nine succeeding years the proportion was only 1 in 8.4; so that not only has scurvy almost disappeared from ships of war and naval hospitals, but the efficiency of the Navy has actually been increased threefold.

[Scurvy, although a disorder of malnutrition, cannot be classed amongst autophagic affections, as a result of general or partial starvation. Men abundantly supplied with the albuminates, the fats, and the carbohydrates, have suffered from it. The cause of scurvy is, probably, to be sought for in a deficiency of the salts, and particularly those salts which are combinations of alkalies with the acids which form carbonates in the system, as the citric, tartaric, acetic, lactic, and malic, whose value and necessity as nutritional agents cannot be doubted. For, setting aside the unproved and unsatisfactory hypothesis of Dr. Garrod, that there is a deficiency of the salts of potash, there is no more evidence to show that we are to look for the cause in the absence of soda, lime, magnesia, iron, sulphur, or phosphorus in the system. Absence of fresh vegetables implies deficiency in the salts of these acids, and scurvy, as we have seen, ensues with certainty on their disuse. "When we inquire," says Dr. Parkes, "whether there is any proof of the deficiency of these particular acids and salts from the diets which cause scurvy, we find the strongest

evidence not only that this is the case, but that their addition to the diet cures scurvy with great certainty. . . . Tartaric and especially citric acid, when combined with alkalies, have always been considered as antiscorbutic remedies, *par excellence*, and the evidence on this point seems very complete."* The writer has found the bitartrate of potash very efficient in the treatment of scorbutic taint, and his experience is supported by the observations of others. Of the vegetable antiscorbutics, the *potato* enjoys, and probably deservedly, the highest reputation; sailors cut it in slices, and pack them in molasses; next to it are *onions*, sliced and eaten raw, which are greedily devoured by scurvy patients. *Cabbage*, in the form of *sourcroul*, *sorrel* (*rumex acetosella*), the *wild artichoke* (MADISON), the *maquey* or *American aloe* (*agave Americana*) (PERRIN), the *prickly pear* (*cactus apuntia*), the *dandelion* (*leontodon taraxacum*)—much used and prized by the French army in the Crimean war—*lamb's-quarter* (*chenopodium album*), *green corn* (SOUTHWORTH, HAMILTON), the *yam*, *apples*—not *cider*—have all been found excellent antiscorbutics. The expressed juice of the *sorghum*, before boiling, was much used in the Southern army, and it is stated, with excellent results. It is unquestionable, that uncooked fruits and vegetables are more efficient than cooked, in the prevention and cure of scurvy. Dried or desiccated vegetables are greatly inferior in antiscorbutic properties to fresh (MARRAIN); and there is always great difficulty in getting soldiers to use them, they having a distaste for them, owing, in a measure, to ignorance of the proper way of preparing them for the table. The writer has seen much benefit from the use of the water in which the desiccated potato had been soaked and boiled, regularly administered to scorbutic men. Fresh or even raw meat, and new milk, are important articles of diet in scurvy, which is ascribed to the lactic acid they contain, an attribute not proved. Heat and light are powerful auxiliaries in the treatment of scurvy; and exposure to the sun's rays, the head being covered, has a salutary effect. In the sclerotic condition of the subcutaneous tissues and muscles, frictions with camphorated lotions, shampooing, alkaline and sulphur baths, have been used with good results. The spongy gums and ulcerated spots should be sponged with solutions of tannin, chloride of iron, or diluted nitrate of silver (BUZZARD). Feter is removed or abated by a wash containing the permanganate of potash. The muriated tincture of iron, with arsenic and quinine, and phosphoric acid, should always be liberally given.

Dr. Parkes advises that the following measures be adopted in time of war, or in prolonged sojourn on board ship, or at stations where fresh vegetables are scarce:

1. The supply of fresh vegetables by all means in our power. Even unripe fruits are better than none, and we must risk a little diarrhoea for the sake of their antiscorbutic properties. In time of war every vegetable should be used which it is safe to use, and when made into soups all are tolerably pleasant to eat.

2. The supply of dried vegetables, especially potatoes, cabbage, and cauliflowers; turnips, parsnips, &c., are perhaps less useful; dried peas and beans are useless. As a matter of precaution these dried vegetables should be issued early in the campaign, but should never supersede the fresh vegetables.

3. Good lemon-juice should be issued daily (1 oz.), and it should be seen that the men take it.

* [Brit. and For. Med.-Chir. Review, 1848. Practical Hygiene, 1866.]

4. Vinegar ($\frac{1}{2}$ oz. to 1 oz. daily) should be issued with the rations, and used in the cooking.

5. Citrates, tartrates, lactates, and malates of potash, should be issued in bulk, and used as drinks, or added to the food. The easiest mode of issuing these salts would be to have packets containing enough for one mess of twelve men, and to instruct the men how important it is to place them in the soups or stews. Possibly they might be mixed with the salt, and issued merely as salt. (*Pract. Hygiene*, 2d ed., 1866, p. 466).]

PURPURA.

LATIN Eq., *Purpura*; FRENCH Eq., *Purpura*; GERMAN Eq., *Purpura*;
ITALIAN Eq., *Porpora*.

Definition.—A disease in which the blood or the capillary vessels throughout the system, or both, are altered, but the nature of the change is as yet not determined. There is evident constitutional disturbance throughout the disease, manifest by disorder of the digestive, the assimilative, and the excretory functions, attended by languor and debility; but it is a disease not usually attended by fever. The capillaries of the mucous and cutaneous surfaces are more especially implicated. Small round spots are visible on various parts of the surface, of a dull red or a deep purple color. These are due to ecchymosis from the cutaneous capillaries beneath the cuticle through which the color shines, and are of small size, except where they run together in patches. Hemorrhages from mucous membranes are common, and also extravasations of blood within the substance of the several viscera.

Pathology.—By some this disease has been considered identical with scurvy, and been named the "land scurvy." Others have considered it simply a disease of the skin—the *purpura simplex* of Willan and Bateman. Authors generally have spoken of the disease under the name of *petechiæ sine febre*, because the spots are not connected with continued fever. The disease is very closely related to hemorrhages as a morbid state on the one hand, and to scurvy on the other; and therefore there may be distinguished two varieties—(a.) *simple* and (b.) *hemorrhagic*, which latter is thus defined,—“The disease when accompanied by hemorrhage from a mucous surface.”

It has not been determined what is the nature of the morbid state essential to purpura. In some cases the urine has been observed to contain an excess of albuminous matter, with a deficiency of urea (COMBE).* The blood has been found deficient in coagulating power. An examination of the blood in two cases of purpura, by Dr. Parkes, showed that, with a somewhat diminished proportion of the solid constituents in general, there was a remarkable increase in that of iron. A peculiar source of danger attends this disease in the occurrence of extravasation of blood into the internal organs. The lungs, the brain, the liver, and the alimentary canal, are the most frequently affected. Purple spots and effusions of blood are to be

* An interesting case may also be referred to, related by Dr. Goodfellow in his admirable lectures on *Disease of the Kidney*, p. 74.

found on the serous membranes, as in the *arachnoid*, the *pleuræ*, the *pericardium*, the *peritoneum*; and occasionally the blood lies coagulated in the cavities of these membranes. It has also been found in the bladder and in the calyces and pelvis of the kidney (CRAIGIE).

Instances occur in which blood oozes, or is discharged more or less copiously, from the mucous membranes without affection of the skin; and to this class of cases, as a form of disease now under consideration, are to be referred those cases of profuse or fatal hemorrhage from slight causes, recorded under the name of "*hemorrhæa*," which have been ascribed to a *diathesis* termed the *hemorrhagic*, and which is supposed to be hereditary (*Edin. Med. and Surg. Journal*, vol. xxv). When the disease is protracted the patient becomes of a sallow complexion, waxy-colored, and dingy; anasarcaous swellings, gangrenous and bed sores appear on the feet and legs, general anasarca prevails, and he dies exhausted. Dr. Craigie considers that the appearance of purple spots in such cases is connected with more or less disease of the heart, especially with hypertrophy or dilatation of the ventricles. The following observation by Dr. Watson puts the pathology of purpura in an interesting and practical light:

"I have adverted to one peculiar source of danger in purpura, the hazard that blood may be effused in some vital organ, where even a slight amount of hemorrhage suffices to extinguish life. Dr. Bateman states that he had seen three instances in which persons were carried off, while affected with purpura, by hemorrhage into the lungs. During the course of one week, in the year 1825, I was present at two inspections in the dead-house of St. Bartholomew's Hospital, illustrative of the same point in respect to another vital organ, and involving a question in forensic medicine. The subjects of examination were both of them women of middle age, who had been brought into the hospital covered with purple spots and bruise-like discoloration, and suffering hemorrhage from the mucous membranes. Each of these women declared that the apparent bruises were marks of beatings received from her husband. One of them became suddenly hemiplegic a little while before she died. Of the manner of dissolution in the other case I am not sure. In both instances a considerable quantity of blood was spread over the surface of the brain between its membranes; and in one of them blood had been shed also into the cerebral substance, which it had extensively lacerated.

"It may be worth mentioning that in one of these corpses there were indications either of unusually rapid putrefaction after death, or (what I think more probable) of some degree of decomposition even before life was extinct. This woman died in the evening, and the body was examined next day, twelve or fourteen hours afterwards. A quantity of fetid gas escaped from the cavity of the abdomen as soon as it was opened, and small bubbles of air were seen to ooze from the areolar tissue of various parts of the body. Even when incisions were made into the *liver*, air frothed up, as it might do under ordinary circumstances from a section of the lungs" (*Lectures*, vol. ii, p. 866).

Symptoms.—Various symptoms, denoting general disorder of the constitution, precede the appearance of the petechiæ, generally by some weeks, such as languor, which is oppressive, weariness, faint-

ness, and gnawing pains at the pit of the stomach. The appetite is variable, generally weak, but sometimes there is an inordinate craving for food, which, when eaten, is said to lie with a weight upon the stomach. The tongue is yellowish, and coated with a viscid fur, the countenance is sallow or dingy, or has a bloated, pale appearance, with swelling underneath the eyelids. The purple spots appear first on the legs, and afterwards, without any certain order, on the thighs, arms, and trunk of the body, and their presence is attended with much weakness and great depression of spirits. No degree of pressure alters the color of the spots, and they are distinguished from flea-bites by the absence of a central puncture. At first the spots are bright red, but in a day or two they become purple, afterwards brown, and when they are about to disappear they assume a yellowish tint. When the disease continues for a long time, all these varieties of color may be seen on a patient at the same time.

The pulse is feeble, and generally a good deal quicker than natural. Deep-seated pains are felt about the epigastric region, as well as in the chest, loins, or belly. In some instances giddiness and lightness of the head prevail, especially when attempting to move or to stand erect, and there may be even dull pain in some part of the head. Constipation of the bowels, palpitation and irregular action of the heart, with a tendency to frequent syncope, are the most distressing and dangerous symptoms.

Causes.—They are not well known. It is a disease more common in women than in men; and, in boys, before than after puberty. It has appeared under the long use of a poor diet, much sedentary occupation, watching, mental distress, and anxiety. It has also appeared under very opposite conditions; for example, in persons accustomed to the use of nutritious food and free living, but in whom there is reason to believe the digestive functions and the constitutional powers have been impaired by the persistent abuse of spirits or malt liquors.

Diagnosis is generally between *Purpura* and *Scurvy*. The following are the principal distinguishing features of each contrasted: (1.) Scurvy is most common late in the winter or early in the spring; purpura in the first seasons of summer and autumn. (2.) In scurvy the gums are uniformly soft, sore, spongy, swollen, and hemorrhagic: this is no common feature in purpura. (3.) In scurvy tumors of a painful kind form in the extremities, with stiffness or contractions of the joints: such symptoms do not attend purpura. (4.) Scurvy is marked by extreme debility, and is rendered worse by bloodletting and by mercury, and is cured by the administration of lemon-juice: not so with purpura, which may require bloodletting for its cure, is not benefited by antiscorbutic remedies, and is sometimes speedily cleared away upon the supervention of mercurial salivation and hypercatharsis (Watson and Wood).

Dr. Graves, of Dublin, has described an *exanthema hæmorrhagicum* in which febrile symptoms ushered in the malady, with an exceedingly dry and brown tongue, and a pulse which conveyed the sensation of small, sharp vibrations with each beat—a kind of wiry

trembling, with an extraordinary *thrill* and *hardness*, "yielding only to the withering influence of approaching death." Bleeding from the intestines and urinary system quickly supervened. These phenomena continued to spread till the whole mucous system of the patient became the seat of copious hemorrhage. A peculiar exanthematous eruption appeared upon the skin, lasting for several days (five), and never presenting any signs of extravasation. Death followed in about four weeks. The pulse did not exceed its natural frequency (70), and there was no disturbance of the nervous system, such as headache, delirium, or want of sleep; and in the beginning of the disease the strength was not remarkably impaired; but subsequently yielded to loss of blood. The disease seemed almost exclusively confined to the arterial and capillary systems of vessels.

Treatment.—To treat this disease with success, it is necessary to ascertain the circumstances under which it becomes developed in each particular case. *Quinine* or *bark*, stimulants, *mineral acids*, *nutritious food*, and *wine*, was the treatment adopted by Cullen, Duncan, and Willan. Subsequently this plan of treatment was questioned by Dr. Parry, of Bath, who found that in his cases a full bleeding from the arm was a much more speedy mode of curing the disease. Dr. Harty, of Dublin, confirmed this principle of treatment, and was successful with liberal doses of purgatives, so as to clear out the intestinal canal completely. *Oil of turpentine*, administered in moderate and repeated doses, has also been recommended. Dr. Hardy, of Dublin, recommends the tincture of *larch bark*. He has long used it as a styptic and carminative tonic; and it is "one of the most elegant forms of prescribing a terebinthinate" (MOORE). *Fifteen-drop doses* of the tincture may be administered every hour, or *eight or ten drops three times a day*, afterwards increasing the dose according to the age of the patient and the necessities of the case.

The treatment of purpura may be comprised in the following measures: The bowels ought invariably, and without exception, to be first thoroughly and effectually evacuated by means of senna, aloetics, or calomel and jalap. If several effectual doses of either or all of these medicines be not followed by less heat of skin, diminution of the frequency of the pulse, abatement of the internal pains, and a cleaner state of the tongue; if the spots continue to increase in number and size, and the hemorrhagic oozings do not cease,—blood, according to age, strength, and other circumstances, may be drawn from the arm, while the patient should abstain from animal food in every form, and should subsist on boiled rice with whey, or the light subacid fruits, as grapes, oranges, strawberries, gooseberries, baked apples, or the like. His drink may consist of tamarind-water, or water acidulated with sulphuric acid. Under this plan most cases of the disease will be speedily and readily brought to a favorable termination. If symptoms of local uneasiness continue after the urgent phenomena have disappeared, leeches should be applied in the neighborhood of the part; and it will be proper to continue the periodical and regular evacuation of the alimentary canal. When the spots have disappeared and the hemor-

rhage has ceased, the constitution recruits rapidly under the gradual but cautious use of light soups and fresh fruits and vegetables (CRAIGIE).

ERGOTISM.

LATIN EQ., *Morbus cerealis*; FRENCH EQ., *Ergotisme*; GERMAN EQ., *Mutterkornbrand*; ITALIAN EQ., *Ergotismo*.

Definition.—A train of morbid phenomena produced by the slow and cumulative action of a specific poison in a fungus peculiar to wheat and rye, and which gives rise to convulsions, gangrene of the extremities, and death, or to symptoms of ill-health.

Historical Notice and Pathology.—Ergotism is a disease very little known in this country; but it is not unfrequent that diseased, unripe, or damaged grain of any kind, and especially rye, is observed to be injurious to the animal economy. Wheat, rice, and such-like grains, are equally injurious when similarly unsound; and the most frequent form of this unsoundness consists in the development of a fungus (the *Ergotaria abortifaciens* or cockspur) upon the grain, to which the name of *ergot* has been given. In this ergot fungus there is a large proportion of fixed oil (WIGGER). A morbid state is said to be produced by various other poisonous fungi, such as the *Amanita muscaria* and *citruria*; the *Hypophyllum sanguineum*; and by plants such as the *lolium temulentum* being mingled with the grain. A most characteristic phenomenon which results is a form of gangrene which follows as a specific effect of the poison in parts remote from the source of circulation. It is a species of mortification which has not been much seen in this country, but is well known, and has been frequently observed, in different parts of Europe, particularly in France, in some districts of which it has been repeatedly known to prevail as an endemic disease, where rye forms the principal food of the inhabitants. The *ergot* or cockspur in rye is only apt to occur after very rainy or moist seasons. In this disease the grains of rye grow to a large size, acquire a black color, and have a compact, horny consistence. The attention of the public was first called to the peculiar mortification which follows the use of this diseased rye by M. Dodard, in the *Journal des Savans* for 1876. He described the appearance of the affected parts, and mentions that fowls fed with the grain soon died. In 1694 its frequent occurrence is noticed at Sologne, and it prevailed amongst the patients of the Hôtel Dieu of Orleans. The upper and lower extremities of those suffering from the disease "grew as dry as touchwood and as emaciated as Egyptian mummies." In 1710 it was again the subject of treatment in the Hôtel Dieu at Orleans, from its extreme prevalence in the neighborhood. About fifty people, men and children, were admitted that season into the hospital. During thirty-three years the endemic appeared three or four times, and always in those rainy or moist seasons in which the rye contained a large proportion of cockspur—more than a fourth. The mortification always began in the toes, and extended gradually along the foot and leg, till it

sometimes rose to the upper part of the thigh. In some patients the gangrened part came away of its own accord; in others it became necessary to assist nature by amputation. In some instances death succeeded to amputation, the disease having continued to extend to the trunk of the body. It is particularly mentioned in a report on the subject to the Royal Academy of Sciences at Paris, that the rye of Sologne, in the year 1709, contained fully *one-fourth* part of the cockspur. The disease generally began in one or both feet, with pain, redness, and a sensation of heat as burning as the fire. At the end of some days these symptoms ceased as quickly as they had come on, when the sensation of extreme heat was changed to cold. The part affected became black, like a piece of charcoal, and as dry as if it had been passed through the fire. A line of separation tended to form between the dead and the living parts, like that which appears in the separation of a slough produced by the application of the cautery.

The disease prevailed in Switzerland in 1709 and 1716, and is described by Langius, of Lucerne. M. Garroud, a physician of Dauphiny, where the disease prevailed in 1709, makes some very important observations on the different symptoms apt to predominate in different individuals. Some patients suffered very violent pain, with an insufferable sensation of heat, although the part felt cold to the touch. In other patients redness, with much swelling, supervened, attended by fever and delirium. The separation of the dead parts from the living took place with excruciating pain. The gangrene was not in every instance dry. Animals were found to die of the specific gangrene when forced to swallow the diseased rye, for they refused to eat food containing it.

The experiments and observations of Tessier show that a given quantity is required to produce the specific effects, and that the action of the poison is cumulative.

The history of some cases of mortification of the limbs related by Dr. Charlton Woolaston, in the *Philosophical Transactions* for 1762, shows that it may occur in this country from eating wheat diseased similarly to the rye; and Sir William Wilde, of Dublin, has recorded its occasional occurrence in Ireland.

Symptoms.—The gangrenous form of ergotism is ushered in by excessive lassitude, more or less protracted, and accompanied with fever; the extremities become painful, cold, and rigid, benumbed, and almost insensible, and are with difficulty capable of movement. Severe internal pains of the limbs prevail (acrodynia), greatly aggravated by heat. It extends by degrees from the toes to the legs and thighs, and from the fingers to the arms and shoulders, when sphacelus supervenes. With the exception of slight febrile heat, the constitutional disturbance appears to be slight, and in this respect resembles scurvy. Ricker has recently described the early symptoms of a case of poisoning by bread containing *ergot*. A family of six persons partook of the bread, and all suffered from the same symptoms—namely, dryness of the throat, epigastric oppression, nauseous taste, mucous and biliary vomiting, vertigo, stupor, and diarrhœa (*New Syden. Society Year-Book*, 1861). Seeing that symp-

toms vary greatly in severity, probably in proportion to the amount of diseased food taken, and the poisonous nature of the particular fungus which affects it, we may have the expression of some phenomena more fully than others—for example, *acrodynia*. Under this name Chomel described a painful affection of the wrists and ankles which prevailed in Paris in 1827, 1828, and 1829. It was then so prevalent among the soldiers in the barracks of Lourcine, that 560 men were affected out of 700; and in that of Comtille, 200 men out of 500. Since 1828 no case has been recorded in the barracks or military hospitals (PARKES). In 1859 M. Barudel observed three cases in the military prison at Lyons; and in spite of negative results of the examination of the quality of the food, Dr. Parkes is inclined to believe that the cause lay there, and probably in ergotism of the flour (*Army Medical Department—Sanitary Report for 1860*, p. 358).

The general train of symptoms produced by the use of diseased grain assumes two forms—namely, the spasmodic or the gangrenous. The spasmodic form commences with a sense of tingling or itching in the feet, followed by cardialgia and similar tingling sensations in the hands and head. Violent contractions of the hands and feet follow, which seem to affect each particular joint, and the pain is said to resemble that of a dislocation. The sensations are also sometimes described as of a bruising kind; and the body is bathed in copious sweats. The symptoms intermit during intervals of two or three days of a remission at one time. Drowsiness, giddiness, indistinctness of vision, and an irregular gait are constant phenomena. Coma and epileptic convulsions are apt to supervene, which generally indicate a fatal result. An enormous appetite accompanies this train of evils. Spots like those of purpura appear on the face, and the disease rarely abates before the third week.

Treatment.—Considerable differences of opinion prevail regarding the treatment of this *dietic* disease. The cause, in the first instance, must be ascertained and removed. To obviate the effects it has already produced, the constitutional treatment must be directed to improve the state of the blood. Tonics and stimulants are to be administered after a free employment of evacuant remedies to clear out the alimentary canal completely. The chlorates of potash and of soda, with antispasmodics, tonics, and narcotics, are especially indicated. *Camphor*, *musk*, *ammonia*, *capsicum*, may be particularly mentioned; and the strength of the patient is to be supported by light, nourishing, and wholesome food.

DELIRIUM TREMENS.*

LATIN Eq., *Delirium alcoholicum*; FRENCH Eq., —; GERMAN Eq., *Delirium tremens*—SYD., *Säuferwahn sinn*; ITALIAN Eq., *Delirium tremens*.

Definition.—*A train of morbid phenomena, produced by the slow and cumulative action of alcohol, in the various forms in which it is used as*

* [The term *Delirium Tremens* was first proposed by Dr. Sutton (*Tract on Delirium Tremens*, London, 1813).—EDITOR.]

a drink. *Delirium* is one of the most prominent features of the morbid state, which is otherwise characterized by hallucinations, dread, tremors of the tendons and muscles of the hands and limbs, watchfulness, absence of sleep, great frequency of pulse. A thick, creamy fur loads the tongue, and a cool, humid, or perspiring surface prevails; while the patient gives forth a peculiar odor, of a saccharo-alcoholic description, more or less strong.

Pathology.—This disease has only been recognized and described since the beginning of this century. The *Experimental Inquiry* of Dr. John Percy, in 1839, illustrating the physiological action of alcohol; an inquiry into the *Physiology of Temperance*, by Dr. Carpenter; the recent *Pathological Observations on the Bodies of Known Drunkards*, by Drs. Roesch and Francis Ogston (1855); and, lastly, a most able and interesting review on the "Treatment of Delirium Tremens," in the *Brit. and For. Med.-Chir. Review* for October, 1859, are contributions which have placed on a more sure foundation our previous theoretical information regarding morbid states which follow the persistent use of alcohol.

The term *alcoholism** is used to denote various symptoms of disease attending morbid processes of various kinds which are capable of being traced to the use of stimulants containing alcohol. The immediate effects of *intemperance* in the use of alcoholic fluids, the nature of *delirium tremens*, and of *spontaneous combustion*, may be embraced under the general designation of *alcoholism*. This term is used in the sense analogous to that in which we use the terms *mercurialism*, *ergotism*, *narcotism*, and the like;—the agents inducing these specific states acting after the manner of a cumulative poison. The progress of modern science has distinctly demonstrated the poisonous action of alcohol; and an account of the nature of delirium tremens, as well as the grounds on which its treatment must be based, are now alike founded on this knowledge. Tiedemann and Gmelin in 1820, and Magendie in 1823, detected alcohol by its odor in the blood. The fluid found in the ventricles of the brain had also been observed to have the smell, the taste, and the inflammability of gin (SIR A. CARLISLE). In 1828 it was theoretically advanced by Leovèille that *delirium tremens* consisted in an exalted state of the vital powers of the brain, excited by molecules saturated with alcohol absorbed from the surface of the stomach and bowels, and carried into the current of the circulation. Now it is a matter of fact, determined by direct experiment, as well as by casual observation, that alcohol is absorbed directly into the circulation, and is capable of acting as a direct poison upon the nervous tissue through which the infected blood circulates. Alcohol has been found in the blood, in the urine, in the bile, in the fluid of the serous membranes, in the brain-matter, and in the liver (PERCY, OGSTON). Its odors can be easily detected in the breath, and the habitual immoderate drinker exhales a distinct alcoholic and saccha-

* [The term *Alcoholism* was first used by Requin (*Éléments de Pathologie Médicale*, t. iii, January, 1852). In the same year Professor Magnus Huss, of Stockholm, published his work, *Die Chronische Alkohols Krankheit*.]

rine odor more or less strong. His clothes at last acquire a spirituous aroma, every part of his body being long thoroughly imbued with alcohol (CRAIGIE). This odor is generally so well expressed in cases of *delirium tremens* that a place has been given to a statement of the facts amongst the characters embodied in the definition. Dr. Percy's experiments directly support these statements, and prove at the same time the great rapidity with which alcohol passes into the current of the circulation. He injected strong alcohol into the stomachs of dogs; and within two minutes after completing the injection, their respiratory and cardiac movements ceased; the stomach was found nearly empty after death, whilst the blood was highly charged with alcohol.

I once had an opportunity of examining the body of a person who for many years had been in the habit of drinking daily a large quantity of brandy. He died of *typhoid asthenia*, with characteristic degeneration of nearly every important organ of the body and of the bloodvessels. The fluid collected from the cavities of the brain, consisting of serum and some blood, contained 2.6 per cent. by volume, and 2.1 per cent. by weight, of alcohol. This quantitative analysis was made for me by my friend Dr. F. S. B. F. de Chaumont, with Giessler's vaporimeter.

The pernicious effects of the continuous use of alcoholic stimuli on the organs and tissues of the body have been deduced from a careful study of the morbid appearances, of a *chronic kind*, met with in the bodies of individuals known to have lived intemperate lives, and who had perished suddenly from the effects of accident, suicide, or homicide, and while apparently in ordinary health and activity. The extent of the chronic changes in the various organs of these individuals is found to have been far in excess of what could have been reasonably looked for in a like number of persons of the same age, and of temperate habits, suddenly cut off while apparently in average health and vigor. The *cumulative* effects of long-continued intemperance have been clearly proved by Dr. Ogston's observations; and the results of his post-mortem inspections, on the whole, support the conclusions which have been arrived at on *theoretical grounds* as to the injurious effects of alcohol in excess. The following statements contain a summary of these results: (1.) *The nervous centres* present the greatest amount of morbid change, the morbid appearances within the head extending over 92 per cent. of those examined. By this observation the theoretical remarks of Leoville, Craigie, and Carpenter are clearly established. (2.) *The changes in respiratory organs* succeed in frequency those of the nervous centres, yielding a percentage of 63.24 of those examined. (3.) *Morbid changes in the liver* are next in order of frequency, and are due to enlargement, granular degeneration, the nutmeg-like congestion, and, lastly, the fatty state. (4.) Next to changes in the liver come those in the *heart and large arteries*. (5.) Next are those of the *kidneys*. (6.) Least frequent of all are morbid changes in the *alimentary canal*.

Two orders of changes may be observed to result from intemperance in the use of alcoholic fluids—namely, one set of long duration, or which at least must have taken some considerable time

before they could be completed; another set of shorter duration, and which probably are more closely connected with the immediate symptoms which precede the fatal event.

The abnormal changes in the cranium, the substance of the brain, its convolutions and cerebral ventricles, all indicate the prolonged action of a morbid poison. The prolonged action of the alcoholic poison on the cranial contents is to produce induration of the cerebral and cerebellar substance in by far the largest number of cases, coincident with an increased amount of subarachnoid serum; while the steatomatous degeneration of the small arteries leads to *atrophy* of the convolutions and *œdema* of the brain.

When spirituous liquors are introduced into the stomach they tend to coagulate, in the first instance, all albuminous articles of food or fluid with which they come in contact: as an irritant, they stimulate the glandular secretions from the mucous membrane, and ultimately lead to permanent congestion of the vessels, to spurious melanotic deposit in the mucous tissue, and to thickening of the gastric substance. By the veins and absorbents of the stomach the alcohol mixes with the blood, and immediately acts as a stimulant to all the viscera with which it is brought in contact. The functions of the brain are at once stimulated, and ideas follow in more rapid succession; the liver is excited to secrete an excess of sugar, by the immediate action of the stimulant on its tissue (Drs. HARLEY and BERNARD). The flow of urine is excited in a similar manner.

In these effects it is impossible not to recognize the operation of an agent most pernicious in its ultimate results, when indulged in habitual excess; but most valuable as a remedial agent, when its action is understood and appreciated (consult Dr. Anstie's valuable work on *Stimulants and Narcotics; their Mutual Relations*). The mere coagulation of the albuminous articles of food and fluid is very different from that effected by the gastric fluids, and tends to render the articles more difficult of solution by the gastric juice.

In these facts it is impossible not to recognize that alcohol being absorbed, a double series of morbid results ensue. On the one hand, a train of phenomena are induced partly of a *chemical* nature and partly *physiological* or *vital*. The general nutrition of the body suffers; and a bad state of health is at last induced, of a peculiar kind, sometimes described as the "*drunkard's cachexia* or *dyscrasia*." This state of the system is characterized by positive irritation, which very soon succeeds to the intemperate use of alcohol, and which is manifested in a variety of ways; sometimes by an unnaturally voracious appetite; but those who over-indulge in the use of such stimuli subsequently suffer a total disrelish for food; they become unable to eat, and dyspeptic symptoms of various kinds betray the irritable state of the alimentary canal, such as stomach-ache, the frequent generation of gases, water-brash, heartburn, squeanishness, vomiting, and palpitations of the heart. A constipated condition of the intestines, attended with deficiency in the power to expel their contents, is very soon established, and sometimes ascribed to the deficient secretion of bile, which is known not to be secreted in due

quantity; and there is every reason to believe, although the fact is not proved, that its quality is deteriorated. Its functional agency on the food and fluids in the intestines is therefore diminished.

If we follow the course of alcoholic absorption through the vascular and pulmonary system, it is found unquestionably to retard the motion of the blood, while it produces a temporary increase in the action of the heart, and a congestion of the whole system of the pulmonary capillary vessels. Respiration is thus, in the first instance, rendered from four to six times more frequent per minute than it otherwise is; and various symptoms of accumulation of blood within the chest, and pulmonary congestion especially, are apt to occur. A short, tickling cough is a most constant phenomenon. Dr. Craigie remarks that all the spirit-drinkers whom he has ever seen or known have been either subject to chronic cough or dyspnoea, or have labored under chronic, dry bronchial disorder, with asthma. That the use of spirituous liquors ultimately retards the motion of the blood in the vessels is known by experiments on the lower animals, and by pathological observations. Thus far we have a morbid condition induced which is highly favorable to the accumulation of fat in the blood, and such an accumulation has been proved to take place. It has been shown by Becquerel and Rodier that fat increases in the blood, in most acute diseases, when the biliary secretion is retarded, and when a scanty amount of food is taken. These are the very conditions which obtain in alcoholism. It is in cases of undoubted drunkenness that fats have occurred in the blood in such obvious quantity as to leave no doubt of its presence. In such cases a milky character is imparted to the serum—a condition which may be recognized by simple inspection; while a microscopic examination and treatment with ether will establish the diagnosis, and distinguish the fat from colorless blood-corpuscles, or from molecules of albumen. Slighter degrees of this condition are altogether physiological, and are met with during the period of digestion, after eating substances rich in fat (A. BUCHANAN, VOGEL). But the extreme degrees of this condition have been especially met with in drunkards. J. Frank regards the white and fatty blood as having its origin in the abuse of alcoholic drinks (*Hannoversche Annalen*, 1847, p. 283, quoted by Vogel). Dr. Adams (*Trans. Med. and Phys. Society of Calcutta*) mentions the case of a sergeant at Fort William who went to bed drunk, and was found dead in the morning. The vessels of the brain were greatly distended with blood, and oil was seen floating in it. Rayer relates the case of a man who, after drinking largely of punch, destroyed himself by the fumes of charcoal. The blood and the urine contained globules of oil. Serules, of Strasbourg, records a similar phenomena. Thus, it is shown by abundant testimony that the blood becomes surcharged with unchanged and unused material, and contains at least thirty per cent. more of carbon than in the normal state. The order of events by which this state comes about is somewhat as follows: Alcohol is directly absorbed by the bloodvessels without undergoing any change or decomposition. Part of it is

eliminated very slowly as alcohol by the lungs, by the liver, and by the kidneys; but appears to tarry in largest amount in the liver and in the brain (PARKES). Another portion is decomposed. Its hydrogen enters into combination with oxygen to form water, which, with acetic acid, having been produced, carbonic acid and water are formed. Oxygen is thus diverted from its proper function, the exhalation of carbonic acid at the lungs is diminished both absolutely and relatively, and less urea is excreted by the kidneys than is consistent with health; but the pulmonary aqueous vapor is not lessened (BÖCKER and HAMMOND, quoted by PARKES). The water of the urine is diminished, the chlorine is greatly lessened, as well as the acids and bases. All the evidence, therefore, points to the effect of alcohol as causing the retention of substances which ought to be eliminated; and this retention of the effete matter is still more intensified by the stimulant action of alcohol increasing for a limited time the frequency of functional acts, followed as it is by a corresponding depression. In this way impaired health is soon brought about, tending to wasting of the tissues generally; and so long as any alcohol remains in the blood as *alcohol*, a certain toxic or poisonous effect continues to be produced upon the nervous system through which the poisoned blood circulates. If a constant supply of the alcohol is kept up, the phenomena of *alcoholism* becomes chronic or persistent; and acute paroxysms, generally in the form of *delirium tremens*, supervene, which is at once the most common and the most prominent evidence of *alcoholism*. In other instances the degeneration of several vital organs generally may become so excessive that death follows by *asthenia*, or with typhoid phenomena ending in coma. [See *Chronic Alcoholism*, by the Editor, vol. ii.]

When mixed with blood out of the body, spirituous liquors cause more or less coagulation, according to their strength and concentration; and when applied to the bloodvessels in the transparent parts of animals, they can be seen to produce the same effects. The congestion that constantly exists in the mucous membranes of the lungs and stomach is evidence of the retarded motion of the blood. The fact that hemorrhoidal swellings are always aggravated by the use of alcoholic fluids is the result of retarded motion of the blood in the hemorrhoidal vessels. When death occurs from poisonous doses, either in animals or in man, although the dose is at first followed by increased frequency of the pulse, yet in a short time the pulse becomes rapid and small; while the extremities become cold, and the power of generating heat is suspended in proportion as the blood progresses slowly and more slowly through the pulmonary capillaries. These effects upon the lungs must be regarded as of a toxic kind; and this specific toxic action is not less obvious on the brain. Its nerve-substance becomes poisonously affected—a condition which seems to constitute one of the most necessary antecedents in the causation of *delirium tremens*.

The effects produced on the medulla oblongata tend to sustain this toxic effect upon the lungs. The brain and the lungs in this respect act and react on each other. Death may ensue in various

ways, but generally by coma, asphyxia, syncope, exhaustion, or epilepsy.

In the case of habitual spirit-drinkers there is thus constantly going on a temporary stimulus and quickened motion of the blood through the vessels, especially manifested by cerebral, thoracic, and hemorrhoidal phenomena, followed by a corresponding depression and tendency to stagnation of the blood in the capillaries of all the internal organs, especially in the membranes and the lax areolar tissues of dependent parts.* The essential nature of *delirium tremens* is associated with the loss of cerebral power, evinced especially in the want of control over thoughts, emotions, and muscular action, consequent on the direct influence of the alcoholic poison. Disturbances of function, depression, and debility are the attendant phenomena. The feeble but rapid action of the heart, the tremulous, undecided action of the muscles, and the terror-stricken and agitated mental state, betoken the depressed condition of the living functions. The amount of phosphates in the urine, as determined by Dr. Bence Jones, is diminished, while the proportion of the sulphates and of the urea is greatly increased. The nature of the morbid lesions found in such cases has been already indicated. The disease has been variously named the *brain fever of drunkards*, *dipsomania*, or *delirium tremens*, by which latter name it is more frequently known and described in this country.

Symptoms and Course.—The disease has been said to become developed under two sets of circumstances—namely, according as the patient has been continuing his potations, or after he has suddenly abandoned them. But its occurrence under this latter circumstance is now known to be nothing more than a coincidence; and of all the errors in popular acceptance connected with the malady, none is greater than that which affirms the exciting cause of a paroxysm of *delirium tremens* to be a sudden stopping or withdrawal of the accustomed quantity of stimulants. The ceasing to drink depends on the commencement of the disease (GAIRDNER). There are some who are constantly taking small quantities of spirits, and who, although they never get completely intoxicated, yet sometimes exceed considerably their accustomed allowance, and continue to do so for some time. The symptoms of *delirium tremens* generally appear in them from the second to the eighth or ninth day after a protracted debauch, and are by some pathologists divided into three stages. The first stage, according to Dr. Blake, is marked by a peculiar slowness of the pulse, by coldness and clamminess of the hands and feet, by general debility, by nausea and vomiting in the morning, and by frightful dreams at night. Very moderate exertion of body causes the patient to perspire profusely, and anything which suddenly affects his mind throws him into a tremulous agitation. The tongue is tremulous and furred, the hands shake, and he is greatly depressed in spirits, sighs frequently, is anxious about

* The influence of alcoholism in causing progressively degeneration of the individual and of the race has been considered at page 144.

his affairs, and is either restless or watchful. These symptoms last from twenty-four to forty-eight hours.

The second stage commences by a hurried and anxious manner, by great excitability of temper, by a small, accelerated pulse; some heat, perhaps, of the surface of the trunk, but accompanied with the coldness and clamminess of the extremities. The tongue is sometimes clean, but often brown and dry, and the patient delirious, suffering from various mental illusions and alienations. In general the delirium is melancholy, and has reference to his usual occupation and habits, or to some difficulty in his domestic affairs. He sometimes sees flames or hears voices talking to him, or as soon as he shuts his eyes he sees people passing under the bed-clothes. In short, he sees objects and sights in situations in which they are not, and which have no real existence; or betrays the most dreadful alarm at hideous objects which he imagines are threatening him with immediate destruction. Restless and sleepless, he moves his trembling hands horizontally over the bed-clothes, as if seeking for something. In general he is harmless and easily controlled; but in some instances he is violent, mischievous, and requires to be restrained. This stage generally lasts from three to four days to a week, when the third stage commences by the patient falling into a sound sleep and gradually recovering, or a fatal collapse comes on, which finally and shortly closes the scene. Without reference to stages of the disease, the following is a general description of its symptoms.

There is always more or less derangement in several other functions besides the brain. The patient is generally void of all appetite; or he may be squeamish, and vomit at intervals. Sometimes he is thirsty, and calls loudly for liquor of various kinds; but often he is indifferent to the sensation of thirst. In several instances great aversion, and even dread, of all food and drink has been evinced; and it has been impossible to persuade the patient to partake of either. The tongue is at first covered with moist white, gray, or slate-colored fur, and when protruded it is tremulous. The bowels are constipated, and less sensible than in the state of health to the action of medicine. When they are acted upon by remedies, the discharges are very dark-colored, the first generally consistent, the latter liquid, dark, and offensive. There are generally fulness and distension, and not unfrequently tenderness and pain, in the epigastric, umbilical, and right hypochondriac regions; and sometimes the two hypochondriac regions give the patient the sensation as if they were drawn tightly together. The skin is bathed about the head and neck with a clammy, unctuous, cold moisture; but elsewhere, and especially at the feet, it is cold, dry, and imper-spirable.

The pulse varies from 96 to 110 or 120, sometimes 130; and though sometimes small and oppressed, is often full, voluminous, and throbbing. The carotid and temporal arteries beat most violently; those of the wrist less forcibly; and the anterior and posterior tibial arteries pulsate feebly enough. The action of the heart

is in general unusually violent, and the cardiac beat is diffused over the whole chest. The respiration is occasionally panting and irregular but not otherwise morbid. Restlessness is extreme. The patient is in constant agitation of mind and body; speaks almost incessantly, yet seldom adheres above a minute to one subject, and is constantly changing place and looking for some new object. He cannot sleep and dreads to be left alone, from the apprehension of spectral visitations. With this restlessness the upper extremities, and especially the hands, are in constant tremulous motion, such that they cannot be kept for two seconds in the same position, nor can the pulse in many cases be accurately numbered at the wrist. Though this tremulous motion of the arms, wrists, and hands is very general, it is not constant; and instances of the mental disorder, agitation, spectral illusions, and sleeplessness have been observed to take place without any such kind of tremors. They are very rarely, almost never, seen in the young or middle-aged to any very great extent, or those whose muscular motions are not otherwise unsteady; and are seldom well marked in first attacks of the disease. Such tremors are principally observed in the cases of confirmed dram-drinkers, whose motions are always unsteady in the morning and early part of the day, until they take a certain proportion of their habitual stimulus (CRAIGIE).

After symptoms of restlessness and sleeplessness have continued for three or four days, the patient may fall into a sound, unbroken slumber, which lasts for some hours. The paroxysm thus works itself out in a definite time in uncomplicated cases; and sleep occurs simply as the natural, the favorable termination of the disorder. It occurs as the *result* of the paroxysm having run its course, and of the nervous system having lapsed into an improved condition, and must not be regarded as the *cause* of those favorable conditions (Reviewer in *Brit. and For. Med.-Chir. Review*, l. c.). On the other hand, the symptoms may pass into a state of *coma vigil*, with constant muttering delirium, *subsultus tendinum*, and picking of the bed-clothes, the pupils become contracted, the muscles of the face and jaw are moved incessantly, and death may ensue from prolonged coma or convulsions. The duration of the paroxysm varies from three, four, or seven days, and a favorable or fatal termination may be looked for in from three to five days.

Diagnosis.—*Delirium tremens* is to be distinguished from typhus fever, and from paralysis agitans, by the previous history of the case, and by the symptoms.

Prognosis.—It is hardly determined what is the proportion of recoveries to deaths; but unquestionably three persons out of four do well. A paper in *The Indian Annals of Medical Science* for 1855, by Dr. Macpherson, notices the great discrepancy in the statistics of writers on *delirium tremens*, with regard to its frequency in both sexes, and to the mortality of the disease. He attributes this chiefly to a want of due classification. Calmeil states the rate of mortality at 5 per cent., Bougard at 19 per cent. The most accurate records to be got are those regarding the British troops at different stations.

The late Sir Alexander Tulloch, in his report for 1853, gives the following percentages of mortality among them:

Great Britain, infantry,	17.6
" cavalry,	18.8
Bermuda,	15.0
Canada,	7.94
Gibraltar,	18.6
Malta,	8.8
Nova Scotia,	9.1

A return of admissions and deaths from *delirium tremens* and *ebrietas* in the General Hospital in Calcutta, from 1848 to 1852, and another of admissions and deaths from the same causes in the Medical College Hospital, during 1851, 1852, and 1853, give some important results, as follow:

That *delirium tremens* occurs in women and men in the proportion of one to twenty-five; but that this difference is due to the difference of habits rather than of sex.

That in regard to age the ratio is as follows:

	Cases.	Deaths.	Per cent. of Deaths.
Ages from 20 to 25,	84	4	9.1
" 25 to 30,	66	16	24.2
" 30 to 35,	48	11	22.9
" 35 to 40,	76	7	9.2
" 40 to 45,	62	6	9.6
" 45 to 50,	28	4	17.3
" 50 to 60,	7	—	—
" 60 to 65,	5	1	—

The greatest mortality is between the ages of twenty-five and forty, which is confirmed by the analysis of another series of sixty-four fatal cases. The percentage shows that there is no uniformity in the proportion of deaths to the number of cases.

There is no evidence to show that the season of the year exerts a definite influence on the occurrence of the disease, whereas the mortality very palpably varies with the temperature—it being more than double in the eight hot months as compared with the four cold months.

The apparent cause of death was as follows: Thirty-three by exhaustion (often with coma); eighteen by coma; eleven by fits (probably apoplectic, called sometimes epileptic); one died on the night-stool; one was found dead in bed.

Convulsions occurred in at least twenty of the above cases. One distinct case of paroxysmal *opisthotonos* occurred in a musician, who during the intervals was able to sit up and whistle tunes.

Treatment.—From the nature of the disease as now described, as well as from the dire results of experience, it is now clearly established that the indications for treatment are,—(1.) The elimination of the poison; (2.) The sustenance of the patient during this period. The two most fatal errors which can be committed in the treatment of *delirium tremens* are either to *bleed* the patient or to give him *opiates*. The greatest number of cases of those treated by opiates

are apt to terminate by convulsions and coma (MOREHEAD, PEDDIE, LAW, CAHILL, LAYCOCK). If it be true, also, that opium and alcoholic stimulants singly are to be deprecated in the treatment of *delirium tremens*, à fortiori in their combination there is a twofold danger; and alike in tropical as in temperate regions it is a course of treatment attended with much hazard, and which, when systematically followed, is certain of leading to unfortunate results (see Morehead's *Researches on Diseases in India*; also, "Notes on Treatment of Delirium Tremens," by W. Hanbury, 33d Regiment, in *Madras Quarterly Journal*, July, 1863).

The strength must be supported by diet of the most nutritious kind, in a fluid and mild form, such as yolk of egg, soups, and the like: food should be given in small quantities, and often. Beef-tea, spiced soup, and egg-flip, are each to be commended at different periods of the day.

Arrangements should be made so that the patient does not catch cold; and if he continues to digest food, the danger is much diminished. The danger in the first instance is from exhaustion; and careful nursing is above all things necessary, so that protection may be adequate and the food adapted to the state of the digestion, which is always feeble. The disease must be treated as one spontaneously curable; not by withholding remedies, but by using them in strict subordination to good nursing and carefully adjusted diet and regimen (WARE, HOOD, PEDDIE, LAYCOCK, W. T. GAIRDNER). Active specifics for *delirium tremens* appear to be founded on the idea that the disease is one nominally of high mortality. Hence the enormous doses of *digitalis* (JONES) and of *Cayenne pepper* (KINNEAR); but before resorting to the use of such remedies as *digitalis*, it will at least be judicious practice to adopt such means as are calculated to restore the powers of nature—namely, nutrients and rest; while the stimulants of such a spice as *Cayenne pepper*, given in the soup, on the atonic stomach, will have a favorable influence on absorption.

In some cases purgative remedies are indicated from the first. "These cases are known by the flushed, bloated appearance, the very foul tongue, the *mawkish*, peculiar odor of the breath, the fetid discharges from the bowels, and the history of a recent surfeit of eating as well as drinking" (W. T. Gairdner, *Clinical Medicine*, p. 271).

Opium may be administered with safety and advantage only in protracted cases, provided the quantity given in twenty-four hours is never allowed to exceed the full dose which would be considered safe for a healthy person of the age and sex of the patient. Where it appears to be indicated in protracted cases, it ought to be pushed as rapidly as possible for two or three doses, while its effects are carefully watched. Its use must be discontinued for at least a good many hours, as soon as a full maximum amount of ʒiiss. to ʒij of the tincture in all has been reached, or even sooner if the pupils have become at all considerably contracted during its use. This remedy should always be given in the fluid form, otherwise it is apt to accumulate in the bowels, owing to the weakened state of the diges-

tion; and a laxative, or even a purgative, should be alternated with opium, followed by a bitter tonic, which always operates favorably in lingering cases of nervous and dyspeptic exhaustion. Narcotics are thus only safe in *delirium tremens* when they are given with the object of aiding and seconding the natural cure of the disease, employed in moderate doses, and given only at the later stages. The heroic use of them, as heretofore too often advocated even by the most eminent physicians, is now recognized as a treatment which merely substituted narcotic poisoning for alcoholism or *delirium tremens*.

LEAD PALSY.

LATIN Eq., *Paralysis ex plumbo*; FRENCH Eq., *Paralysie saturnine*; GERMAN Eq., *Beilähmung*; ITALIAN Eq., *Paralisi litargirosa*.

Definition.—A series of morbid phenomena induced by the absorption of the salts of lead contained in solution in drinking-waters, or in various foods and drinks, or conveyed into the system through the integuments of those who are in the habit of handling the soluble salts of lead; or through the pulmonary mucous membrane of those exposed to the influence of vapors containing lead.

Pathology.—The theory of this disease is, that the lead being absorbed, produces a peculiarly painful affection of the alimentary canal, termed *lead colic*, or *painters' colic* (eq., *colum ex plumbo*). It may also affect the muscles of the extremities, producing palsy; and finally it produces ulceration of the gums and alveolar processes, accompanied by a peculiar blue line, which was first pointed out by Dr. Burton, of St. Thomas's Hospital. This blue line is seen along the free margin of the gums, but is absent where a tooth or stump is wanting. To this the name of "blue gum" has been given. A stain, also from lead, sometimes affects the conjunctiva.

The fact of the lead being absorbed and mingled with the blood is demonstrated by the circumstance that lead has been obtained from the coats of the stomach of a dog poisoned by lead, even as late as a month after poisoning. Again, MM. Duvergie and Guibourt have detected lead in the brain of the human subject, and Dr. Budd has detected it, not only in the human brain, but also in the muscles. Many pathologists are inclined to believe that the blue line observed in the gums of persons poisoned by lead is owing to the presence of lead in some peculiar state of combination, as with some of the constituents of the tartar of the teeth (TOMES); and from the observations of the late Dr. George Wilson, it appears that there are various tissues of the body for which lead has an affinity, and that it is more apt to be found in some organs than in others. The stomach and cæcum of a pony that died a fortnight after being removed from the sources whence lead was received into its body through the ingesta, having been carefully analyzed by this able chemist, it was found that, while the contents of these viscera did not contain the metal, the substance of their tissues

yielded it in small but manifest quantity. In a mare dying under similar conditions the following organs were examined: (1.) A part of the lungs; (2.) A part of the heart; (3.) A part of the large intestine and contents; (4.) A part of the stomach and duodenum; (5.) The spleen; (6.) One kidney; (7.) A portion of the liver. The spleen yielded the most abundant and most deeply colored precipitates; while the intestinal canal gave the faintest indications. Next to the spleen the liver yielded most, afterwards the lungs, then the kidney, next the heart, and least the intestines.

The general result of these analyses went to show,—*First*, That in cases of slow poisoning with lead, the metal comes to be diffused through the entire body, and exerts its poisonous action, though in an unequal degree, on every organ; and *Second*, That lead having once entered the body, leaves it again very slowly, so that long after an animal has ceased to receive lead in its food or drink, or by any other medium, we may expect to find the metal in its tissues; and the restoration to health is always protracted. The lead passes off by the urine sometimes in large quantities, but very slowly (PARKES). Fletcher found in the urine of a man with lead colic no less than 4.8 grains of metallic lead in 100 grains of solid matter of the urine (*Dublin Med. Press*, 1848); and Dr. Parkes records a case in which the last exposure to the influence of lead was on the 20th December, 1852, and lead was found in the urine before treatment commenced on the 16th June, 1853, and the blue line was still perceptible below the edge of the lower gum.

Colica Pictonum rarely causes death. De Haen opened many persons that died from lead poisoning, and found in all a constriction of the colon, and in a certain number a similar affection of the cæcum. Merat opened seven cases, and observed a similar appearance. Dubois de Rochfort says that he found in two cases intussusception of the intestines; but Andral examined five cases, Louis one case, and Martin another, without finding any morbid appearance.

Mr. Hunter had an opportunity of examining the state of the muscles of the palsied hand and arm of a painter who died of a broken thigh in St. George's Hospital, and found them all of a cream color, being probably in a condition of fatty degeneration. In the paralysis arising from the poison of lead the tissues of the muscles and nerves are early affected, and at a subsequent period the nervous centres become implicated. The muscles seem to be first affected, and through them the nerves participate in the contamination, which gradually advances to the nervous centres in the severer forms of the disease. This is shown by the fact that local paralysis always precedes, and generally for some considerable time, those phenomena which indicate disease of the nervous centres, and which show that contamination of the system has been great. The evidence now is also abundant which proves that it is lead existing in the affected tissues which thus contaminates and impairs their function. The morbid appearances in the brain and spinal cord are generally such as denote imperfect or depraved nutrition of those centres. The brain is pale and soft, and its convolutions wasted, the

sulci between them wide, and sometimes patches of white softening are to be seen in the hemispheres; and this seems to be more particularly the case in those who have had paroxysms of an epileptic nature, and in whose brains lead has been detected (TODD).

The introduction of lead into the system has taken place in a great variety of ways. In France the pernicious effects were wont to occur from putting a lump of litharge into the *vin gâté*, to cover its acidity and render it salable; and from this having been practised to a great extent by the Pictones, or the inhabitants of Poitou, the disease has been named *Colica Pictonum*. In the cider counties of Great Britain this disease formerly existed to a great extent, and has been termed Devonshire colic, or *Colica Damnoniensis*. The impregnation of cider with lead in this country was generally the effect of accident, and arose from the troughs in which the apples were crushed having the different pieces of stone of which they are composed clamped together with iron, and fixed by melted lead. In some districts it was the practice to line the entire press with lead, or to tip them with that metal. It was a custom, also, almost universal, to make the upper part of the boiling vessel of lead; while some growers, in managing weak ciders, put a leaden weight in the cask to sweeten the liquor. From these and perhaps other causes, Sir George Baker found the cider he examined to contain four and a half grains of lead in eighteen bottles, or a quarter of a grain in each bottle. In the West Indies lead poisoning appears to have been produced by using leaden worms to the stills, by which the rum became impregnated with this metal. There are many other minor sources of poisoning by lead; as keeping pickles or preserves in glazed earthen vessels, and coloring confections with preparations of lead. The still and other machinery used in the distillation of fermented liquors being now constructed of metals so combined as not to be acted upon by acid fruits or sugar, diseases from the action of lead are no longer so common as they were wont to be, but are confined principally to laborers in the lead manufactories and to painters. The use of paint where lead exists is the most common source of its absorption in this country; and hence house painters are those most frequently affected. The paint called "flatting" (or that which is mixed with a large amount of turpentine, so as to give a *flat, dead, or non-glistening* surface) is the most injurious to the workman. The turpentine, readily passing off by evaporation, carries with it a small supply of lead, which is constantly and gradually inhaled, or it is left on the skin to be absorbed, or mixing with saliva, it gets into the stomach. By one or all of these ways the system becomes affected, first through the circulation of the blood, and subsequently by the constituent tissues of the organs combining, in some form or other, with lead, which is thus deposited in them.

All ages, both sexes, and all classes are liable to the poisonous action of this metal; but the workers in lead have been at all times the greatest sufferers. Women in this country often suffer from lead colic, but it is rare to find them paralytic; men suffer both from the colic and the characteristic palsy.

Symptoms.—The quantity of lead necessary to produce its specific results, or the time it takes to accumulate in the system when introduced, is not determined; and both the dose and the time varies greatly in different individuals. Sometimes all its most pernicious effects are produced by one dose taken by the mouth; and then again, if introduced by the skin, months, and even years, may elapse before the system is laid under its influence. As a general rule, however, a much smaller dose will produce colic than is necessary to produce palsy.

When the dose is of such intensity as to produce *colica Pictorum*, the symptoms do not differ, except in being of greater intensity, from those which mark ordinary colic. There is the same dragging and twisting pain, and the same relief by pressure; the same absence of fever; the same unhurried pulse; the same constipation, only more obstinate; and, in the worst cases, the same vomiting. Andral, who treated upwards of 500 cases at La Charité in the course of eight years, says it is not strictly true that the pain in lead colic is always diminished on pressure; for in the greater number of cases pressure neither augments nor diminishes the pain, while in some cases the sufferings of the patient are increased by it. He also says it is as common to find the abdomen distended with gas as to find it drawn in, and the *rectus* muscle strongly contracted on both sides. The symptoms peculiar to this form of colic are, occasionally an attack of epilepsy, and an ulcerated state of the mouth, accompanied by a blue line on the dental edge of the gum—a discovery which the profession owes to the careful observation of Dr. Burton.

The duration of the constipation which attends *colica Pictorum* is very various. Three or four days may elapse before a stool is procured, and when the case is early submitted to medical treatment, seldom more than a few hours; but fifteen days have been known to elapse without a stool. As soon as the bowels act, the great severity of the disease is mitigated; every symptom is gradually relieved, and the disease generally terminates within a week.

But when palsy is the result of the absorption of lead, a painful state of the arms often precedes it, as well as repeated attacks of the characteristic *colic*. The nerves of the forearm and hand become first affected, and the affection is in general limited to the upper extremities, the extensor muscles of the hands and fingers become paralyzed, so that when the arms are stretched out, the hands hang down by their own weight, causing what the patients term "*wrist-drop*." The disease may be sometimes seen limited to one finger. More commonly, however, it affects the whole arm, and sometimes so completely that the patient can execute no movement with it; and when lifted up, it falls like an inert mass. Sometimes the extensor muscles of the limb are alone affected; and in this case the hand is often strongly closed by the powerful and unresisted action of the flexors. When the case continues for a few weeks, the posterior surface of the forearms (where the extensor muscles are situated) is rendered quite concave, from atrophy and consequent shrinking of the muscles. The arm loses its plumpness, and even the interosseous membrane may be felt. The muscles of the ball

of the thumb are also in a similarly wasted state. In general both arms are palsied, but not equally so, one being slightly more affected than the other. Supposing both sets of muscles to be equally palsied, the patient usually recovers the use of the *flexors* before that of the *extensors*, so that he can carry a weight hanging in his hand before he can shave himself. This restoration of the lost power is usually accompanied by more or less pain. The duration of the palsy under any treatment is always long, and often lasts many months, and in some cases years. Both colic and palsy may occur an indefinite number of times. When epilepsy is produced, the fit does not differ from epilepsy due to other causes.

Diagnosis.—The colic of lead poisoning can only be distinguished from ordinary colic by the history of the case, and by the blue line on the dental edge of the gums, but which is present only where the teeth or their stumps are in the alveoli, and ceases where a tooth is completely wanting.

The palsy is to be distinguished from cerebral paraplegia by the history of the case, by the integrity of the intellect, and by the blue line on the gum. A most important means of diagnosis in paralytic affections is the electric current properly administered. The excitability of the muscles is always much diminished in paralysis from lead, as Dr. Althaus clearly shows, and often it is entirely lost. Such is the case, not only when the muscles are atrophied, but when the bulk of the muscles is only slightly diminished; and even after the voluntary movements have regained their former power, the excitability of the muscles to the electric current still remains impaired. The relation of the muscles to the stimulus of Faradization helps in doubtful cases to establish the diagnosis, as the excitability of the muscles is always either lost or diminished in lead palsy, whilst it is normal in spontaneous paralysis. *Therefore, when the muscles of a paralytic limb move well under the influence of the electric current, we may fairly conclude that there is no lead in the system* (Althaus *On Paralysis, Neuralgia, &c.*, p. 72).

Prognosis.—The termination of lead colic, except where the dose has been in such excess as to produce death in a few hours, is always favorable; and those cases which prove fatal are generally such as have been exposed to the cumulative influence of lead for a long time, and who have been intemperate.

The palsy does not appear greatly to affect the health of the patient; but in some cases it has hitherto not been cured or relieved. In general, however, the patient recovers, although perhaps not completely. Drs. Garrod and J. W. Begbie have satisfactorily demonstrated that lead poisoning of the system exerts a remarkable influence as a predisposing cause of gout; and my friend, Dr. W. England, of Winchester, as the result of his experience, spontaneously volunteered to me a similar remark. Inveterate forms of dyspepsia may be traced in many cases to the influence of salts of lead in the drinking-water, its pernicious influence expressing itself differently in different constitutions, although never amounting either to colic or to paralysis.

Treatment.—The treatment of *colica Pictonum* is extremely simple. The objects to be obtained are to procure stools, a copious discharge of urine, and perspiration, with a view of eliminating the poison from the body. To allay pain is also an urgent necessity. For these purposes five grains of calomel, fifteen grains of jalap, and one grain of opium, should be administered as soon as the patient is seen; and at the end of two hours about two ounces of *camphor mixture*, combined with a drachm of *sulphate of magnesia*, and twenty minims of *tincture of hyoscyamus* may be given every two or every four hours, till the bowels are freely evacuated, when relief more or less complete is obtained. The mixture should be continued at proper intervals for three, four, or five days, when the patient, though greatly weakened, has in general recovered.

In a few cases, however, the pain continues, and with considerable severity, after the bowels have been freely evacuated. The practice in these instances is to apply a blister to the epigastrium, and to keep the blister open for a few hours; and this additional application will generally complete the cure.

The patient is also much relieved if placed in a warm bath, and at the same time directed to inject repeated enemata of hot water, that stools may be readily obtained. In the absence of the warm bath, a large linseed or mustard poultice should be applied over the abdomen.

With respect to the cure of lead palsy, an endless variety of treatment, both local and general, has been tried, but with so little positive result that, when the patient has recovered, it has been doubtful whether it has been owing to the great length of time that has elapsed, or to the medicines he has been taking. It has been believed that sulphur has the power of neutralizing the effects of lead by forming some innocuous compound with it. It is not known, however, whether any such compound is really formed, but a remedy of a very useful kind exists in the form of a *sulphur bath* (TODD). The ingredients of this bath consist of from two to four ounces of the *sulphuret of potassium*, mixed with from twenty to thirty gallons of water. The *ergot of rye* (*secale cornutum*) has been said to produce a considerable increase in the power of the flexor muscles of the arm in about a fortnight, and the improvement gradually extends to the flexors, till at the end of about three months the patient has recovered. This may have been the natural result of elimination of the lead. The experiments of Orfila long ago rendered it probable that lead is removed from the body by the kidneys; and *iodide of potassium* promotes the elimination of lead in this way. It may be used with advantage combined or not with the *citrate of iron*, the use of iron, in some form or other, having been found of benefit in cases of palsy from lead contamination. Dr. Parkes has chemically proved that lead can be made to pass off by the urine, by the action of *iodide of potassium*, in the same way as mercury is known to be eliminated.

The principle upon which the *iodide of potassium* acts has been pointed out in a most interesting manner by Melsens. He assumes that the lead is in actual union with the affected tissues, being

retained among them as an *insoluble* compound. The *iodide of potassium*, after its absorption into the blood, combines with the lead, and forms with it a new and soluble salt. The poison is thus liberated from its union with the injured part, dissolved out from the damaged fibre, and once more set afloat in the circulation. Thus the poison and the remedy are cast out together by the urine (MELSENS, WILLIAM BUDD). It is necessary, however, to notice the dangerous phenomena which may at first supervene on the administration of *iodide of potassium* in cases of lead poisoning; and great caution is necessary in the employment of this remedy in man for the first few days. At the moment when the metallic compounds fixed in the body become dissolved or transformed, the phenomena of acute poisoning may occur, caused by their liberation. So much is this the case, that the treatment may be supposed to be at first hurtful rather than beneficial. The patient should have beside him a graduated solution of the *iodide of potassium*; and should begin with a small dose (fifteen grains during the twenty-four hours), and afterwards increase or diminish it according to his pains and sensations (MELSENS).

Galvanism, in the form of Faradization, ought to be used as a local stimulant to the nerves, with the precaution that its application is not to be continued too long each time. Ten or fifteen minutes, at three different periods of the day, or of every second day, and persevered in for not less than four weeks, will be found of great service (TODD, ALTHAUS). The beneficial influence will follow, although, in the commencement of the treatment, even a current of very high tension does not cause any movement whatever in the paralyzed muscles. In such cases the beneficial influence seems attributable to the restoration of mobility to the molecules of nerve and muscle by an induced current, and which is necessary to enable them to be physiologically active. Severe shocks, especially in the commencement of the treatment, should therefore be carefully avoided, as by such the weakened excitability of nerve and muscle may be reduced, in place of being fostered and developed (ALTHAUS, *l. c.*, p. 112 and 119). It would be rational, however, to defer the application of galvanism till the lead has been completely eliminated.

GOITRE.

LATIN Eq., *Bronchocèle*; FRENCH Eq., *Gottre*; GERMAN Eq., *Kropf*—Syn., *Struma*; ITALIAN Eq., *Gozzo*.

Definition.—*A specific affection of the thyroid gland, induced by the persistent use of water which has percolated through magnesian limestone rocks or strata, and containing the soluble salts of lime in solution.*

Pathology and Morbid Anatomy.—The characters of the swelling of the thyroid gland, associated with this morbid state, appear to be different at different stages of its existence. At first the tumor is soft, but it gradually acquires a firm and even a cartilaginous consistence. In the soft condition the cell-elements of the gland seem to secrete a fluid of a thick, ropy, viscid, gelatinous appearance; but

when the consistence increases, the hypertrophy of the cell-elements is generally more obvious than the fluid secretion, its bloodvessels seem increased in size and number, and ultimately cysts become developed, in which the glairy fluid abounds. In the more advanced cases, osseous, or at least calcareous deposits occur, and occasionally the whole organ is transformed into an osseous-like capsule, filled with matter of various kinds, which has been likened to jelly, suet, and honey. Sometimes the gland preserves its original lobulated form, its relative proportions being maintained; at other times there is no distinction of parts or lobules. The right lobe is more often enlarged than the left (ALIBERT, RICKWOOD, GREENHOW). Solid aggregations of calcareous particles have been found in the goitrous gland (CEELY.) The tumor, originally of a simple nature, is liable to enlargement of its arteries, to expansion of its cells, or the formation of cysts on its surface and its interior, or to inflammation and ulceration of its component tissues. Such ulceration is apt to assume a malignant scirrhus-like appearance.

The prevalence of the disease is limited to certain districts of peculiar geological formation; and wherever it prevails, popular opinion has always regarded the water used for drinking as being in some way connected with its cause. Alike in England and in Oude, where goitre prevails, the people are convinced that water in some way is the cause of goitre. It was at one time believed that *snow-water* from the summits of lofty mountains contained the poisonous material which established *goitre* among the inhabitants of the valleys. The people of almost all the valleys of Switzerland drink snow-water, or the water of the glaciers; but *goitre* abounds in some of the valleys only; and in Greenland and Lapland, where snow-water is commonly used, *goitre* is unknown. Again, the disease exists in countries in which snow is never seen, as in Sumatra; and in places where snow never lies sufficiently long to be used as a drink, as in Derbyshire. It prevails in places where *pump-water*, rather than *surface-water*, is used. Still, the evidence is very conclusive which points to something specific in the quality of the water as tending to the development of *goitre*. At Nottingham, in England, where this disease prevails, the common people refer it to the *hardness* of the water—i. e., to its impregnation with calcareous salts, *sulphate* or *carbonate of lime*; and it has even been affirmed that the presence of magnesian limestone always implies the endemic coexistence of the disease (INGLIS). In Captain Franklin's expedition to the polar sea, *goitre* was found to be very prevalent at Edmonton, where the soil is calcareous, and contains numerous fragments of magnesian *limestone*. The disorder attacks those only who drink from the *water* of the river (the Saskatchewan). In its worst form it is confined almost entirely to the half-breed women and children who make use of the river-water. The men, who are often from home journeying through the valleys, drink the melted snow, and are less affected; and should incipient symptoms of the disease come on in winter, when they live at home and use the water of the river, the annual summer voyage to the sea-coast generally effects a cure. The natives who use *snow-water* only, and drink from the

rivulets which flow through the plains in summer, are exempt from the disease; but the residence of a single year at Edmonton, if the river-water of the Saskatchewan is used, is sufficient to render a whole family the subjects of goitre (RICHARDSON). The disease has been known to occur and to affect a family in a very short time, who, being free from the disease while using the *surface-water*, had a well dug, and obtained their water by tapping a limestone rock; and after drinking from this well for a short time the disease appeared among them. There are some waters in a goitrous district in Switzerland, issuing from the hollows of certain rocks, and trickling along crevices in the mountains, the drinking of which will produce *goitre*, or augment goitrous swellings, in eight or ten days, while the inhabitants who avoid these waters are free from the disease (BAILY, WATSON). Dr. Coindet, of Geneva, states that the use of the hard pump-water in the lower streets of that town brings on goitre very speedily; and at Cluses, on the Arne, numerous cretins and goitrous persons are seen in the streets; while lofty cliffs of mountain limestone tower over the town; and through the crevices of these cliffs copious streams of water flow. In Yorkshire, Derbyshire, Nottingham, Hants, and Sussex, in England, where the disease prevails, there is a ridge of magnesian limestone running from north to south through the centre of the district. All along that line goitre prevails to its greatest extent; and, diverging to either side, the disease is found to diminish (INGLIS, *Treatise on English Bronchocele*). The disease has been known to prevail in one great section of the province of Kemaon, in India, south of the Himalayan mountains, and to be almost entirely absent from another section of that district. Both of them agree in their external aspect, altitude, and climatology, but differ so remarkably in their geological formation that an examination of the rocks of the district, into the very villages where the disease abounded, or did not abound, enabled one to predict whether the inhabitants were affected with goitre or not. No instance occurred, in a district extending over 1000 square miles, in which goitre prevailed to any extent where the villages were not situated on or close to limestone rocks (McCLELLAND). Dr. McClelland visited 126 villages scattered promiscuously over an area of upwards of 1000 miles. The following are the results he obtained:

1. Five of these villages were built upon hornblende and mica slate, or on siliceous sandstone, or on green sandstone. They contained 290 inhabitants, not one of whom was a *cretin*, or was affected with *goitre*.

2. Seventy-one of the villages in the same district were built upon clay-slate. These contained 3957 inhabitants; and among them there were twenty-two persons with goitre, or *one* in two hundred of the population. There was not a single cretin.

3. Thirty-five of the villages, having a population of 1160, were built upon Alpine limestone; and in them 390 persons, or more than *one-third* of the inhabitants, had goitre, while thirty-four of them were cretins, or about one person in every thirty-five.

Lastly, goitre is extremely frequent at Secrora, near Lucknow, and in all that district of Oude which stretches towards Nepaul and

the Goruckpore district beyond the Gogra. In the jungles of the Teraie, at the base of the Nepaul hills, the disease is very frequent; and in Nepaul itself, among the inhabitants of the *Cis* and *Trans-Himalayan* regions, it is constantly met with (BRAMLEY; GREENHOW; *Indian Annals* for 1857 and 1859). All along the line of Teraie, on to Goruckpore, goitre is so prevalent that one in ten persons is afflicted with the horrible disfigurement. The kingdom of Oude is geologically made up of the diluvial detritus of the Himalayan chain, which abounds in limestone, and the soil of the district contains abundance of lime, which is taken up by the waters that percolate through it from the rivers and from the rains and floods. The lime thus taken up and held in solution with carbonic acid gas is frequently found deposited round nuclear fragments of flints or stones, and is then known by the name of "Kunker;" so that wherever "Kunker" abounds, there soluble salts of lime, silica, alumina, and sometimes magnesia and protoxide of iron, will be found (SLEEMAN, O'SHAUGHNESSY, GREENHOW).

There is a remarkable circumstance connected with the prevalence of goitre in Oude, which is, that it affects animals as well as man. At Hissawpore, a village about twelve miles distant from Secrora, on the Surjoo River, dogs and other animals are affected with it (GREENHOW). Mr. Bramley noticed the same curious fact in Nepaul; for "on one occasion a goat brought forth a kid with a goitre as large as its head. Puppies of a month old, bred from English dogs, are very frequently affected by it, as are also lambs."

A curious outbreak of *acute goitre* occurred in 1860 in the garrison of Briançon (Hautes Alpes). The mean strength of the garrison during the year was forty-eight officers and 954 men; and from this force fifty-three cases of acute goitre (fifty-one soldiers and two *cantants de troupe*) were admitted in the year, and in the first three months of 1861. One case occurred in as short a time as *eight* days after arrival at Briançon, and one after *sixteen* months' stay; but the majority (thirty-nine) occurred after from *eight* to *eleven* months' sojourn. The rapidity of growth of the gland was remarkable. In some very predisposed subjects eight days sufficed to show a large increase; and the form of the tumor was most frequently bilateral. The sanguino-lymphatic temperaments and robust constitutions were those most attacked. There was no obvious hereditary tendency; but several of the men came from departments where goitre is more or less prevalent. The men who came from maritime places, and who were placed at Briançon under quite unusual conditions (4285 feet above the sea-level), suffered most. M. Collin, who records the outbreak, does not say a word about the composition of the water; but the position of Briançon renders it highly probable that the water of the place is charged with lime and magnesian salts (Parkes, in *Army Med. Department Sanitary Report* for 1860, p. 385).

The disease is known to prevail at the base of lofty mountains in many parts of the globe. It is endemic at the foot of the Alps, where it is frequently associated with *cretinism*—a sort of idiocy, associated with atrophy of the brain and deformity of the body. It is also endemic at the foot of the Apennines. It is common in Derby-

shire, where it is called the "Derbyshire Neck." It is met with in some flat situations in Norfolk; and in one village about five miles from Cambridge it is extremely common (WATSON). In South America goitre is met with both in the upper and in the lower course of the Magdalen River, and in the flat high country of Bogota, 6000 feet above the level of the stream (HUMBOLDT). It is also common at the base of the South American Andes. In North America many cases occur in the vicinity of the Blue Ridge, in Virginia. It is prevalent in the mountainous regions of Pennsylvania, New York, New Hampshire, and Vermont (DUNGLISON). In India it prevails in Oude, and along the line of the Himalayan range. It seems to be more common in females than in males, and is rarely seen before the age of puberty; but in districts where the disease abounds, it is on record that children are sometimes born goitrous of goitrous parents (GODELLE, WATSON). The evidence of hereditary transmission, in the strict sense of the term, appears to be doubtful; but predisposition may exist in some, rather than in others, to the development of the disease.

Cretinism.—The condition of idiocy named *cretinism* (and associated with goitre in many districts) is of great interest; but the relations of the two are not yet clearly understood. The idiocy of *cretinism* is associated with deformity and imperfection of the bodily organs, the brain, in common with other parts, participating in the imperfection and deformity. (1.) It may be defined as: "*A condition of imperfect development and deformity of the whole body, especially of the head. It is endemic in the valleys of certain mountainous districts, and is attended by feebleness or absence of the mental faculties and special senses; and is often associated with goitre.*" The affection of the mind varies from mere obtuseness of thought and purpose to the most complete obliteration of all intelligence. (2.) Three varieties are to be distinguished: "(a.) *Complete Cretinism*—Synonym, *Incurable Cretinism*,—*Cretinism characterized by idiocy, deaf-dumbness, deficiency of general sensibility, and absence of the reproductive power.* (b.) *Semi-Cretinism*,—*A degree of cretinism in which the mental faculties are limited to the impressions of the senses and the bodily wants; the general sensibility is obtuse, the head is badly formed and drooping, the speech is rudimentary, and the reproductive powers are feeble or absent.* (c.) *Incomplete Cretinism*—Synonym, *Curable Cretinism*,—*A degree of cretinism in which the mental faculties, though limited, are capable of development; the head is moderately well formed and erect, the special senses, the faculty of speech, and the reproductive powers are present.*" Dr. Guggenbühl, of Zurich, was the first to recognize the fact that the mental state of *cretins* would be improved by improving the growth and condition of the body. In 1842 he succeeded in buying the mountain of Abendberg, which incloses the plain of Interlaken, and there he established an hospital for these unfortunate children. There the infant *cretins*, removed from the low, close valleys (in which the malady too often finds the circumstances most congenial for its development), are fed and trained in "the free, dry, cool and bracing air of the open but sheltered and sunny slopes of the Abendberg." With but few exceptions, *cretins* are goitrous;

and it has been said that when both parents are goitrous for two generations in succession, the offspring, being the third generation, are sure to be cretins (WATSON). The cretin is found chiefly in the valleys of the Pyrenees and the Alps, in the mountains of Syria, in the hilly parts of China, and in the Himalayan regions; but the disease is not confined to the lower valleys of Switzerland, or to those other mountain districts of the Old and New World which resemble it in physical conformation. All over Europe the victims of this disease may be seen; and Virchow found, in his official inquiry into this subject, that no less than 133 decided cases were living in the villages of Lower Franconia; and in Germany, Sweden, Norway, England, and even in London, isolated cases of *cretinism* are to be met with. The stature of the *cretin* is diminutive; his head is of great size, but flattened at the top, and spread out laterally; while the countenance is vacant and void of intelligence. The nose is flat, the lips are thick, and the tongue is large. The skin is dark-colored, coarse, and rough. The abdomen is sunken and pendulous; the legs are short and curved.

Virchow's dissection of the heads of *cretins* has led him to conclude that the primary abnormality of the brain commences with the growth of the bones of the *basis cranii*, and especially with the *sphenoid* and the adjoining parts. In the normal state the basilar part of the occipital bone, the *sphenoid* and *ethmoid*, with their intervening cartilages, form a portion of an arch; while the same parts in a *cretin* are early ossified into the form of a rectangle, early union of the bones taking place, with various lesions of the intervening cartilages. With such early union, arrest of growth occurs at that part of the skull; but various compensatory developments continue in other parts. Hence the *prognathous* face, and the sinking of the root of the nasal bone. Irregular and partial union of the sutures at an early age is a frequent morbid condition of the insane, associated with an atrophic condition of the *gyri* below the site of union. The oblique downward direction of the orbit in *cretins* is brought about by the compensating growth of the skull generally, and more especially of the *malar*, the *frontal*, the *temporal* bones and *zygomatic* arches, in consequence of the deficient development of the *sphenoid* bone. The stunted development of the bones at the base of the skull gives a very short distance between the front and middle part of the cranium; while the diminished growth of the nasal septum and of the jawbones gives a *prognathous* form of face alike to the cretin, the negro, and the monkey.

It may generally, therefore, be concluded, from the cumulative nature of the evidence, that a poison exists in association with lime and magnesia in geological formation, whose action induces undue ossification and thickening of the base of the cranium, tending to diminish the size of the foramina for bloodvessels (KÖLLIKER, VIRCHOW); and it is fair to connect the unusual quantity of lime taken into the system with such premature and abnormal ossifications. Wherever chemical examination of the water used by the inhabitants of the different places where goitre and cretinism prevail has been made (as it has been especially in India), it has always been found

to contain a large quantity of carbonate of lime; whereas the water derived from the clay-slate rock, and which was drunk by the inhabitants who did not suffer from *goitre*, contained none. Such observations as those described, and especially those of McClelland and Greenhow, show that neither the atmosphere, the elevation above the sea-level, the physical aspect of the country, nor locality, have anything to do with the production of *goitre*; but they prove almost to demonstration that the affection is due to some specific action of the drinking-water which flows from rocks of a particular geological formation named magnesian limestone. The circumstances under which these affections were found by McClelland to exist in the low, burning plains of Bengal, formed a striking corroboration to his observations in the hills of Kemaon. Goitre and cretinism are very prevalent in different parts of the district of Goruckpore. The soil of the district is of two sorts. One, to which the natives give the name of *Bhat*, characterizes the lands bordering the river Gunduk and its branches. This soil is remarkable for the large proportion of calcareous matter which it contains. One specimen, on analysis, yielded upwards of twenty-five per cent. of carbonate of lime. Goitre and cretinism are very prevalent in the villages built upon this soil. In some of them ten per cent. of the population are affected; and of the children in the villages where *goitre* prevails ten per cent. are *cretins*. The dogs and cats of these villages are also often affected with the disease. On the other hand, the lands on the banks of the Gogra consist of a soil to which the natives give the name of *Bangar*. It is much less retentive of moisture than the *Bhat* land, and requires irrigation for the production of winter crops. This *Bangar* soil is very siliceous, and contains scarcely any lime. Goitre and cretinism are unknown in the villages built upon this soil (*Brit. and For. Med.-Chir Review*, Jan., 1861).

The natives of Oude ascribe their goitres to drinking certain waters; and they adduce cases to prove that by partaking of the water of certain wells they get the disease, and by deserting those wells they sometimes become cured of it (GREENHOW). Thus almost all writers who have written on the subject agree that, in some way or other, the condition of the water has to do with the production of *goitre*. Remarkable instances are known wherein the exchange of well- for rain-water, for drinking purposes, has been followed by the best effects, and even by the disappearance of goitrous tumors. Dr. Greenhow states that in Oude, where the water of wells believed to be injurious, in consequence of their excessive impregnation with lime, has been given up, and other water used instead for drinking, great benefit has been felt, and goitres have decreased in size, even though the subjects of them have continued living in the same village as before. He was assured also, by several of his patients in Oude, that certain wells were known by them to be deleterious, and that the natives of the villages avoided them accordingly, having learned to do so from experience. He tested the water of the wells most shunned by the natives, and found it to contain a great excess of lime; and he concludes, from his own investigations, in connection with others, that the use of drinking-

water containing lime is the main cause of goitre. How it acts on the system is as yet unknown.

A form of *goitre* totally different from the endemic form has received the name of *exophthalmic goitre*, the *goitre* of *anæmia* or *spanæmia*, and may be defined as "*Enlargement, with vascular turgescence of the thyroid gland, accompanied by protrusion of the eyeballs, anæmia, and palpitation.*" It is rare in children; more common in females than males; and coexists with wasting discharges, or supervenes upon them; such as in leucorrhœa, menorrhagia in females, and hemorrhoids in males. It is sometimes associated with heart disease. The normal nutrition of the nerve-centres is obviously impaired. Sleep is disturbed and unrefreshing. Digestion is impaired. Pallor and anæmia therefore ensue, with palpitation of the heart and carotid arteries. A systolic *bruit* may be heard over the region of the heart, and sometimes over the carotids; and during any excitement a rushing or throbbing feeling is experienced in the head. The thyroid body now seems to act as a diverticulum. Simple hyperæmia prevails in the gland, which subsequently becomes hypertrophied. The exophthalmic state is similarly induced; continued distension of the intraorbital vessels presses the eyeballs forwards; and the exophthalmic state may be reduced by pressure applied to the carotids. In fatal cases the eyes are observed to recede within the orbit after death (BÉGIN). Vision is rarely impaired. Dilatation of the cavities of the heart is the lesion which is induced in that organ, and dilatation of the veins is the last expression of the disease (Fletcher, in *Brit. Med. Journal*, 23d May, 1863).

Treatment of Goitre.—The indications for treatment are,—(1.) Removal from the district where the infection occurs, or the improvement of the water used as a drink; and (2.) The elimination of the poison. As in the case of mercury and of lead, so we find that *iodide of potassium* has a wonderful effect in subduing the swelling, and probably in eliminating the poison which produces goitre, whatever that may be. *Iodine* has thus acquired the reputation of being almost a specific against goitre (COINDET, STRAUB, GAIRDNER, MANSON, LUGOL). In robust subjects it has been advised that the administration of *iodine* should be preceded by general bloodletting, on the principle that the abstraction of blood favors absorption; but this should not be done unless the *iodine* does not seem to be producing any effect; and whatever may be the preparation of *iodine* employed, it must be persevered in for a length of time, and the dose gradually increased (DUNGLISON). The best form of administration is that in which the *iodine* is mixed with and dissolved by *iodide of potassium*. It is then held in solution, and is sometimes known as *Lugol's solution*. It is made by dissolving *one part of iodine* (five grains) with *two parts of iodide of potassium* (ten grains), and adding water (to the extent of *one pint*). It contains *one grain* of iodine in four ounces of the solution. The treatment ought not to commence with a larger dose than *one drachm* three times a day. Small unirritating or alterative doses are the most efficient, and they may be gradually increased when necessary. The remedy

may also be employed externally in the form of a liniment or ointment—the *unguentum iodum compositum*, of which a small portion may be rubbed upon the swelling night and morning. In some parts of India the application of an ointment of the *biniodide of mercury* was found very efficacious. The ointment is prepared by adding finely powdered *biniodide of mercury* to melted lard or mutton suet. This ointment is then applied to the goitre about an hour after sunrise; and is rubbed in, by means of an ivory spatula, for about ten minutes—the patient sitting with his goitre held well up to the rays of the sun as long as he can bear the exposure. The ointment will probably produce a blistering effect, although no vesicles appear on the skin; and in the course of the day the ointment should be gently rubbed in again, and the patient sent home, with orders not to touch it with his hands, but to allow the ointments to be gradually absorbed. A second application is sometimes necessary in very bad cases. In 1855 no less than 500 or 600 persons were sometimes treated in a single day; and it is estimated that altogether about 60,000 patients have been so treated; so that the cases in the district are now far less numerous than formerly; and the disorder is thus being extinguished (Mouat, in *Indian Annals* for April, 1857).

When medical treatment fails, surgeons have attempted to give relief to the symptoms by one of three operations; but so long as the disease does not interfere with any of the important functions of the body, nor produce serious discomfort, surgical interference is not warrantable. These operations are,—(1.) The introduction of setons through the tissue of the diseased gland—an operation which has been successful. A thin double wire is to be passed through the gland, and left there for a week (QUADRI, TANNER, JAMES). (2.) Tying the thyroid arteries which supply the goitre with blood, and so starving the tumor, has been attended with varied success (COATES, BRODIE, EARLE, WICKHAM). These means having failed, (3.) The gland has been extirpated—an operation which few surgeons would now think of undertaking.

Dr. Watson justly observes, regarding these surgical interferences for the radical cure of goitre, that “there is not one of which the average results have been sufficiently prosperous to warrant its repetition, except in cases where life is put in jeopardy, or made miserable by the swelling; and where other methods, and particularly the treatment by *iodine*, have been tried and have failed” (*Principles and Practice of Physic*, vol. i, p. 795). He makes, however, an exception in favor of puncturing any cell or cyst containing fluid, which sometimes makes up a considerable portion of the tumor. Such cysts may be punctured without much risk, and with great relief to the patient.

The indications for treatment in *exophthalmic goitre* are, to allay the irritability of the stomach by the use of ice; to give bland, unstimulating, nutritious food in small quantities and at short intervals; to produce sound and refreshing sleep by morphia, or any such stimulating soporific; to administer digitalis; to steady the weak heart and control its excitement. Iron may improve the

state of the blood ; but the *hygienic* conditions of our lives are mainly to be rectified (FLETCHER, l. c.). Begbie recommends the use of *belladonna* in cases of

PARALYSIS OF THE LOWER LIMBS PRODUCED BY *LATHYRUS SATIVUS*.

Definition.—A specific form of paralysis, comes suddenly by stiffness in the legs about the knees, unsteadiness of gait, till at last paralysis becomes so great that the feet are so dragged upon the ground that, with the legs inwards, and the knees bent, the great toe scrapes the ground. Recovery of ease occurs from the use of the flour of the last of wheat, *avena sativus*; and ill-health is apt to occur when the quantity of flour exceeds one-twelfth part; and if the proportion exceeds one-third, the consequences may be serious.

Pathology.—Attention has recently been called to paralysis by Dr. Irving, civil surgeon of Allahabad, prevalent in part of that district. Village of Allah Barra, on the right bank of the Jumna, and lame persons, whose paralysis is well known to themselves to be due to their having lived on bread made from the flour of the *Lathyrus sativus*, are aware that it has a peculiar effect on the locomotor system (IRVING). From statistics that have been collected it is found that a proportion of 3.19 per cent. of the population is rendered useless by this disease (Court and Irving, 1857). Different villages are affected in different proportions. The country where it prevails is a vast swamp, and it appears that the *L. sativus* is most extensively cultivated as an article of food, enough in most parts of India, and is frequently sown in wheat or barley, and cut down green as fodder for cattle. It is used as food when made into flour, but it is not used in wheat or barley flour; and it is only when it is used in this way that it is injurious; and when it exceeds one-third of the flour, paralysis sets in. Wheat flour will not grow in the country where the natives are in a great measure left to their starchy bean, and suffer in consequence. This is also known in Thibet; and even in Europe the natives follow the use of the *L. sativus* as an article of food. Several species of the same genus are occasionally known to be poisonous (DON, TAYLOR, LOUDON). Cattle, however, fed on the beans, are said to become paralyzed. The use of bread made from the flour of *L. sativus* is known to establish complete paralysis of the locomotor system in a young and healthy man in a few weeks. Sir James Simpson of the same family, who had been in the habit of eating bread made of the flour of *L. sativus*, suffered more or less from similar symptoms, and died in 1847, *Ann. d'Hyg.*, Avril, 1847, p. 469; Taylor,

[Further accounts of this vetch as a cause of paralysis may be found in *Indian Annals of Medical Science*, vol. vii, 1861, by the late Dr. Kinloch Kirk, p. 144; also by Dr. Irving, pp. 127 and 501. An incidental reference is also made in Thomson's *Travels in the Western Himalaya and Tibet*, p. 391, footnote.]

Symptoms and Phenomena.—The paralysis is observed most frequently during the rainy season in India—cold and wet being perhaps an exciting cause, so that the first lameness may be a mixture of palsy and rheumatism. Men who had gone to bed quite well awoke in the morning feeling their legs stiff, especially at the knees, their loins weak, and their gait unsteady. Fever does not seem to attend the accession of the more obvious phenomena; but pain gets worse, and eventually the lower limbs become quite paralyzed. The patient walks with difficulty, the toes turn inwards, the legs waste, and the great toe nail scrapes the ground, till, in persons who go barefooted, the nail has been known to get rubbed down to the quick. Males are said to be more often affected than females; and the *Ryots* are more liable to the disease than the *Zemindars*.

Treatment.—Some cases seem to have been benefited by generous diet, tonics, the use of strychnine, and of blisters to the loins; but nothing is known definitely on the subject, nor have we any records of the morbid state of the spinal marrow in such cases. Of course, the bad quality of the food must be set right.

CHAPTER XI.

PATHOLOGY OF THE PARASITIC ORDER OF ZYMOTIC DISEASES.

The Parasitic order of diseases are so called from the fact that a great variety of lesions and symptoms of organic disorder are brought about by the presence of *animals* or of *plants* which have found a place to live and subsist within or upon some tissue, organ, or surface of the body of man, or of other animals and plants. The diseases of this order may be considered as due either,—(1.) To the existence of parasites from the *animal kingdom*; or, (2.) To parasites from the *vegetable kingdom*; and all of which live either upon some surface or within a cavity of the body, or within the substance of some of its tissues or organs.

From the animal kingdom we have the *entozoa* and the *epizoa*, and from the vegetable kingdom the parasitic diseases are due to *epiphytes* and *entophytes*. It is only recently that we have been able to point with distinctness to a vegetable parasite finding its way actually into the substance of animal tissues, and there progressing in development. Dr. H. V. Carter, the Professor of Anatomy and

Physiology in the Grand Medical College of Bombay, has described a "*fungus disease*" of the foot, in which numerous minute tubercles, resembling fish-roe, lie beneath the muscles," and affect the tissues from the bones to the skin (*Trans. of the Med. and Phys. Society of Bombay*).

Plants, as well as man and animals, have their peculiar parasites and parasitic diseases. The mistletoe is a familiar example of a *vegetable parasite*; and the oak apple, or gall-nut, is a familiar example of an *animal parasite* affecting a plant.

It is known, and in many instances it is capable of experimental proof, that some of these parasitic diseases (vegetable as well as animal) may be transmitted or communicated indifferently from animals to man, and from man to animals. The *tape-worms*, the *encysted*, *vesicular*, and *round worms*, are examples of parasites intercommunicable among animals; and *Tinea*, from the "*Dartre ton-surante*" of the horse, ox, and cat, having been communicated from these animals to man, are instances of vegetable parasites intercommunicable among animals. It may be that the blights of plants, or the causes of them, are also communicable to animals and to man. We know that some of the diseases of man and animals are intimately related with famines and unwholesome food, and that famines are due more to diseases of vegetable and animal life than to destruction or loss of food.

The records of history furnish numerous examples of periods of blight in the vegetable kingdom, associated with epizootics among the lower animals, and with epidemics affecting the human family (see Sir William Wilde's *History of Ireland*, compiled in connection with the census taken ten years ago). The relative connection of these events has scarcely yet attracted the attention of pathologists, human or comparative. Here, indeed, is a wide field for investigation—a territory almost yet unexplored. The medical service of Her Majesty's British and Indian armies gives golden chances for observation, if the chances are seized at the moment, and the observations connected with the facts already known. To the more salient of these facts the attention of the student is here directed.

Since the beginning of the present century, when Rudolphi published his systematic work on the entozoa (1808), almost every year has contributed new and important facts, which render the subject of *Parasitic diseases* one of increasing interest to the pathologist and the physician. The subject abounds with most puzzling riddles in natural history and pathology, especially concerning the reproduction, the development, and the propagation of parasites. So long as 170 years ago (1691), the independent nature of such structures as the "*hydatid cyst*" was established by Tyson (*Phil. Trans.*, cxci, p. 506); and it was stated by Pallas in 1766 that all the cystic worms were forms of tape-worms belonging to one species—namely, the cystic or hydatid tape-worm; but it was not then known how their generation and propagation was effected. For a very long time the received doctrines regarding the generation and de-

velopment of living beings were tacitly set aside in behalf of such "existences." They were believed to arise spontaneously. Inquiry was thus set at rest, curiosity seemed satisfied, or investigations followed a fruitless direction—as when observations were made on such cysts, in the hope of discovering in them some evidence of the existence of organs of generation, or evidence of some process of generation analogous to what prevails in other animals. Ova were looked for, and organs of generation were looked for, where neither ova nor organs of generation existed. The calcareous particles visible in the tissues of those animals were at one time mistaken for eggs, and described as such, in the membrane of the *Cysticercus* (1841). At last, in 1842, a great insight was obtained regarding the nature of the generation and development of these and other parasites by the publication of facts which showed that amongst a certain class of minute *Cercariæ* (worms of a microscopic size found in stagnant water), the generation of them was carried on through a series of broods produced from one parent, each brood differing from the parent and from each other. The discovery of this fact was due to Steenstrup. He described the phenomena under the name of "alternation of generation" amongst these *Cercariæ* which ultimately live within the body of different mollusca (*Planorbis* and *Lymnæus*).

These observations gave quite a new direction and impetus to investigation; and Steenstrup himself foretold that the hydatid cysts would be proved to be undeveloped tape-worms, each cyst capable of producing a tape-worm after its kind.

This view was at once taken up, and independently worked out by Eschricht, Nordmann, Von Siebold, Kuchenmeister, Kræmar, Zenker, Leuckart, Weinland, in Germany; Von Benedin, in Belgium; Dujardin, Blanchard, and Robin, in France.

Many physiologists and physicians of this country have been no less accurate observers. Barker, Bristowe, Nelson, Erasmus Wilson, Gulliver, Gull, Jenner, Busk, Rainy, Cobbold, and Bastian, may be particularly noticed; and many valuable records have been published in isolated papers by officers of the Army Medical Department.

The conjoint researches of these extensive workers have found most philosophical expositors in this country in Dr. E. A. Parkes, the Emeritus Professor of Clinical Medicine in University College, and now Professor of Hygiene in the Army Medical School (*Brit. and For. Med. Review*, 1853); in Dr. Allen Thomson, Professor of Anatomy in the University of Glasgow (*Glasgow Med. Journal*, No. x, July, 1855); and lastly, in Dr. William Brinton, in the *Brit. and For. Med.-Chir. Review* for 1857. From these and many other later sources the following concise account may be given relative to the parasitic order of diseases, and their rational treatment.

Kuchenmeister and Von Siebold were the first to prove by experiment that the hydatid or vesicular worms were the young or larval states of tape-worms; and they demonstrated—(1.) That each parasite had an independent life of its own. (2.) That most animals have each their own peculiar parasites—that even parasitic

animals are themselves infested with parasites—an observation embodied in the Hudibrasian couplet,—

“These fleas have other fleas to bite 'em,
And these fleas, fleas, *ad infinitum*.”

The experiments of Kuchenmeister and Von Siebold further demonstrate—(3.) That some parasites pass or migrate from the body of one animal into that of another (including man), or from one part of the same animal to another cavity or viscus in it; such migrations being required for the introduction of the entozoa or their ova into the animals they inhabit, and where they undergo those series of changes about to be described, by which they reach maturity. (4.) That thus, through food or drink, or both, entozoa pass into the human body, finding their way into the most delicate tissues, as most minute ova or embryos. (5.) That they undergo progressive changes of development towards maturity in each of the new localities where they find subsistence and protection.

We cannot now rest satisfied with a mere knowledge of the general appearance of these so-called “worms” as they are found in man and animals. It behooves the physician to ascertain their origin, their source, and their mode of entrance into the body they inhabit. The easy but unsatisfactory hypothesis of “spontaneous generation” can no longer be entertained. On the contrary, it is now clearly established that all the parasitic entozoa are produced more or less directly from fecundated ova. The general and minute anatomy of these “worms” must be studied, as well as their modes of generation, of reproduction, and phases of progressive development, the various metamorphoses of their individual forms, and their transmigrations from one animal into another. We must become acquainted with their existence even in plants, as well as in animals, and in other animals besides man, especially in such animals or plants as constitute the food of man—fish, flesh, fowl, mollusca, and crustacea,—and especially all fresh-water plants, or plants which grow on moist ground.

A knowledge of details relative to generation and reproduction is absolutely necessary in order to appreciate the nature of parasitic diseases. Indeed, without such knowledge no advance is likely to be made in the prevention of these diseases. It is this kind of knowledge which has recently led to most important practical results in the history of animal parasites, and which most of all seems capable of extending the science of parasitic diseases, especially in relation to human pathology, and the rational treatment of such diseases.

Parasites of animal organization exist in man and animals in every grade of development; and the first lesson for the student to learn is, how to distinguish entozoa which are sexually complete from those parasitical productions which are destitute of sexual organs, but which have long been regarded as distinct animals.

At least thirty well-marked forms of entozoa have been described as infesting the body of man. They may be enumerated in a classified list as follows:

*LIST OF GENERA AND SPECIES OF HELMINTHOID ENTOZOA WHICH HAVE BEEN DISCOVERED INFESTING THE HUMAN BODY.

A. SOLID WORMS: PLATYLMIA; vel, STERELMINTHA.

I. CESTOIDEA—Banded, riband-like, girdled, or tape-worms in the form of—

1. Mature sexual parasites, androgynous, and living in the alimentary canal.

- (a.) *Tæniæ*. 1. *Tænia solium* (LINNÆUS), the common tape-worm of man in this country.
 2. " *mediocanellata* (KUCHENMEISTER), the common tape-worm of man on the Continent.
 3. " *marginata* (BATSCH, COBBOLD).
 4. " *elliptica* (BATSCH, COBBOLD).
 5. " *acanthotrias* (WEINLAND, COBBOLD), its larva, scolex, or cysticercus only known.
 6. " *nana* (SIEBOLD), a very small filiform *Tænia*.
 7. " *flavopunctata* (WEINLAND, COBBOLD).
 8. " *echinococcus* (SIEBOLD).

- (b.) *Bothriocephali*. 1. *Bothriocephalus latus* (BREMSER); vel, *T. lata* (LINNÆUS), the broad tape-worm, endemic to man in some localities only. Its embryo is ciliated and developed in water (KNOCH).
 2. *Bothriocephalus cordatus* (LEUCKART), new to science; recently found in North Greenland.

†2. Immature non-sexual, cystic, or vesicular parasites, the embryonic form of the genera sub (a.) *Tæniæ*.

- (a.) *Cysticerci*. 1. *Cysticercus tæniæ cellulosa* (RUDOLPHI), the larva or scolex of the *T. solium*.
 2. " *tæniæ medicanellatæ* (LEUCKART), the larva or scolex of *T. medicanellata*.
 3. " *tenuicollis* (RUDOLPHI), the larva of *T. marginata*.
 4. " *tæniæ ellipticæ*, at present unknown.
 5. " *tæniæ acanthotrias* (WEINLAND), only the cysticercus found; mature *Tænia* not yet found.
 6. " *tæniæ nanæ*, at present unknown; probably inhabits insects (LEUCKART).
 7. " *tæniæ flavopunctatæ*, also at present unknown.

- (b.) *Echinococci*. 8. *Echinococcus hominis* (RUDOLPHI), the larva of *Tænia echinococcus*.

II. TREMATODA—Fluke-like parasites.

1. *Fasciola hepatica* (LINNÆUS); vel, *Distoma hepaticum* (RUDOLPHI).
 2. *Distoma crassum* (BUSK); *Distoma Buskii* (LANKESTER).
 3. *Distoma lanceolatum* (MEHLIS).
 4. *Distoma ophthalmobium* (DIESING).
 5. *Distoma heterophyes* (SIEBOLD).
 6. *Bilharzia hæmatobia* (COBBOLD); vel, *Gynacophorus hæmatobius* (DIESING).
 7. *Tetrastoma renale* (DELLA CHIAJE).
 8. *Hexathyridium pinguicola* (TREUTLER).
 9. *Hexathyridium venarum* (TREUTLER).

B. HOLLOW WORMS: NEMATELMIA; vel, CØRLELMINTHA.

I. ASCARIDES—Unisexual, body attenuated posteriorly, and still more so anteriorly, mouth with three tubercles, tail of the male narrower than that of the female.

1. *Ascaris lumbricoides* (LINNÆUS).
 2. *Ascaris mystax* (RUDOLPHI, COBBOLD); vel, *Ascaris alata* (BELLINGHAM).
 3. *Trichocephalus dispar* (RUDOLPHI).

* The individual names in this list are similar to those published by Dr. Cobbold in a number of the *Lancet*, before the publication of the second edition of this work. In that list a number of forms were introduced for the first time by Dr. Cobbold, which had never before been noticed by any of our systematic writers; and Dr. Cobbold took great pains in working out this point. His name, therefore, appears to identify them in the list. (See also his great work *On Parasites*, recently published.)

† N.E.—The appearance produced in the flesh of animals by the growth of these vesicular parasites has been named "the measles," or "measly flesh."

- II. OXYURIDES—Unisexual, body more attenuated posteriorly than anteriorly, rudimentary tubercles round the mouth, tail of male, thickened.
 1. *Oxyuris vermicularis* (BREMSER).
- III. TRICHINÆ—Unisexual, cystic, and free.
 1. *Trichina spiralis* (OWEN).
- IV. SCLEROSTOMA—Unisexual, body slightly attenuated anteriorly, mouth with four hooks, tail of male, cup-shaped.
 1. *Sclerostoma duodenale* (COBBOLD); vel, *Ancylostoma duodenale* (SIEBOLD).
- V. STRONGYLUS—Unisexual, body attenuated posteriorly, mouth with six lobes, tail of male, cup-shaped.
 1. *Strongylus bronchialis* (COBBOLD); vel, *Filaria bronchialis* (RUDOLPHI).
 2. *Eustrongylus gigas* (DIESING); vel, *Strongylus renalis* (MOQUIN-TANDON).
- VI. SPEROTTERA—Unisexual, tail spiral, and furnished with marginal appendices.
 1. *Sperottera hominis* (RUDOLPHI).
- VII. FILARIE—Unisexual, body equal (filiform), mouth with three tubercles, tail simple.
 1. *Filaria lentis* (DIESING); vel, *Filaria oculi-humani* (NORDMANN).
 2. *Filaria medinensis* (GMELIN).
- C. A third class may provisionally be regarded as accidental Parasites. These are—
Pentastoma constrictum (lung, liver; West Coast of Africa).
Anthomia caricularis, larva, exciting causes of boils.
Pentastoma denticulatum vestris hominis (SIEBOLD), liver and small intestines.

The sexually mature entozoa inhabit either the alimentary canal of animals or the cavities of the lungs; or to express it generally, they inhabit such parts of the body as are in immediate or free communication with the external air. On the other hand, the non-sexual or immature entozoa, while parasitic, all live inclosed in cysts; such cysts being situated either in the parenchyma of organs, or in close internal cavities—*e. g.*, the eye within secreting tubes, bloodvessels, and the like. In such places these non-sexual parasites are all proved to be incomplete animals. They are the embryos, larvæ, or early forms of entozoa, which only attain to sexual maturity by migration from the place of their earlier abode into the alimentary canal, or pulmonary or other open cavity of different animals. Or leaving their encysted parasitic state in the condition of larvæ, they reach maturity in a free state, when they are developed in water, in earth, mud, or upon moist plants, or in other conditions favorable for them.

The cystic or vesicular entozoa, established by Rudolphi as a separate order of parasites, are to be distinguished from those which are not vesicular, but which are also inclosed in cysts. Some of the early parasitic forms of round worms, as well as others, are thus inclosed in cysts (*Trichinæ spiralis*). All entozoa so encysted are found to be immature; and in no instance has the encysted entozoön been known to attain sexual completeness, however well grown it may appear to be, so long as it remains inclosed in a cyst. Sometimes cysts only are found, which may be identified as pathologically altered conditions of cystic or encysted entozoa. When free, all these entozoa come at last to acquire sexual organs, and, when they have arrived at maturity, to exercise the function of sexual reproduction. The number of fecundated ova which most of them produce is enormous. In a tape-worm, or *ascaris*, there are many millions; but "the struggle for existence" consigns the greater part of them to death as the food of animals unfavorable to their growth as parasites.

The process of fecundation and the development of the embryo

from the ovum have now been actually observed in a considerable number of the parasitic entozoa; and it is to be remembered, as a general fact, that the development of the ova rarely takes place in the same animal, or in the same part of an animal, where the parasitic entozoön has passed its life and has exercised the generative function. There is either a migration from a parasitic to a free condition for a time (e. g., *Guinea worm*, *Ascarides*, *Cercariæ*); or from one animal into another animal, the free condition intervening (e. g., *Bothriocephalus*); or, lastly, the migration may take place from one part to another of the same animal who is the unfortunate host (e. g., *T. spiralis*; and cases of tape-worm giving rise to *Cysticercus*). Some entozoa, known only as incomplete or immature animals in the parasitic mode of life, attain to sexual maturity in the free state; others again, and perhaps the greater number, after living free for a time, become sexually complete in the parasitic condition (e. g., the *Ascarides* and the *Bothriocephalus*).

"The migrations or changes of habitation of the entozoa, or their ova or embryos, appear to take place in a variety of ways: *first*, by their being passed out of the body of the inhabited animal with the fæces or other secretions; *second*, by their being introduced into the bodies of inhabited animals with their food or drink; *third*, by their directly piercing the integument or other tissues, *fourth*, by their piercing the membranes and parenchyma, entering the bloodvessels, being distributed through them, and subsequently piercing their coats to attain other situations.

"Some of these entozoa are directly developed from their ova, without undergoing more remarkable changes than those which are known usually to accompany the process of embryonic evolution in many other animals. Other entozoa are subject to individual metamorphoses, or the embryo passes through successive stages of development, of so remarkable a character as to mask the regular sequence of the phenomena of progressive formation. There are others of the entozoa which are subject to still greater changes in the progress of their existence,—changes upon which great light has recently been thrown by the remarkable researches of Steenstrup and others, in regard to what has been called alternate generation or *metagenesis*. Thus some of the entozoa, by a non-sexual process, undergo that peculiar form of multiplication in which the immediate progeny of development from the ovum is dissimilar from the parent, but produces, without the aid of sexual organs, another progeny, which either itself, or by repetition of an analogous process, returns to the parental form. This is a process of the nature of an internal or external gemmation, which is often attended with a prodigious multiplication of the number of individuals. In some entozoa, again, metamorphosis and metagenesis are combined. It is obvious that the external conditions necessary to maintain these varieties of the vital states must be different" (Allen Thomson, *Glasgow Med. Journal*, l. c.).

The lesions and diseases caused by the existence of parasites rather tend to embitter existence than to cause death; and they are especially frequent amongst soldiers. With one exception—namely, in the case of the immature cystic parasites—the disorders

induced are, as a rule, not severe; indeed, it is a condition of parasitism that it should not actually destroy the life of the animal from which it derives its own subsistence.

It is the immature parasites which tend to destroy the life of their host by the lesions they induce, and the destruction of parts which they cause, when they pass from one place to another, or from one state to another onwards to maturity. Thousands of mature worms infest children, yet they do not appear ill. But such is not always the innocent history even of the mature worms,—undefined illnesses, violent and sudden pains, febrile phenomena like typhoid or rheumatic fever, chronic inflammations, wastings, convulsions, chorea, epilepsy, amaurosis, apoplexy, giddiness (staggers in sheep and horses), are the grave results which sometimes befall human or other animals who may become the unfortunate hosts of such undesirable guests as mature or immature encysted parasites. Sometimes even death is the result.

Dr. Heslop, of Birmingham (*Dublin Quar. Jour.*, No. 55), says:

"1. That in the great majority of cases of tape-worm, and, though with lesser frequency, in cases of other intestinal worms, more or less serious and peculiar nervous disturbances are apt to arise.

"2. That the most frequent of these are headache, giddiness, various troubles of the special senses, especially singing in the ears, flashes and dark spots before the eyes, imperfect amaurosis, and trembling of the limbs.

"3. That various anæsthetic and, on the contrary, neuralgic phenomena are very frequent, usually connected with general lassitude and sense of muscular feebleness.

"4. That, though less frequent than those previously cited, convulsive seizures, partaking of the nature of epilepsy or acute eclampsia, or sudden attacks of insensibility, mixed with syncope and, in the female sex, severe forms of hysteria, are also often directly traceable to worms.

"5. That the last symptoms (No. 4) are more common in childhood and the earlier periods of life than afterwards, and are more frequently caused by the round and thread-worms than by tape-worm.

"6. That chorea does not appear to be often excited by the irritation of worms.

"7. That a feeble state of the general health generally accompanies the presence of worms; often in cases of tænia, proceeding to marked anemia, so as even to lead to the suspicion of the possible existence of Bright's disease.

"8. That the irritative phenomena of the digestive tube, even when associated with various symptoms referred to the functions of that tract, do not warrant the diagnosis of the presence of tænia; and that their absence does not absolutely indicate the absence of the parasite.

"9. That the frequent appearance of the nervous symptoms above related, without a well-marked relation to any special lesion of the nervous system, especially if alternating with periods of perfect or nearly perfect freedom, should engender the suspicion that worms are present. If to these symptoms are added various ill-defined disturbances of the functions of assimilation, including occasionally colicky pains, without marked vomiting, pain after food, or decided emaciation, it is in the highest degree probable that worms are the source of the symptoms.

"10. That it is probable that many of the symptomatic phenomena of

vermination are connected, not with their direct irritation of the mucous membrane with which they are in relation, but with a general disorder of the system, partly resulting from the parasites, and partly the cause of their maintenance and development in the intestinal tract."

CHAPTER XII.

DETAILED DESCRIPTION OF THE PARASITES, AND OF THE LESIONS ASSOCIATED WITH THEM.

SECTION I.—THE ENTOZOA.

TAPE-WORMS—*Cestoiden*.

Definition.—*In their mature condition the tape-worms are all more or less jointed entozoa, of a riband-like form, marked with bands, or girdled. Each mature joint or segment is of hermaphrodite conformation, containing at once male and female reproductive organs, which produce fecundated ova. In their immature condition the embryo penetrates the tissues, and becomes encysted. In this stage of development they are known as the "cystic entozoa."*

Pathology.—Eight varieties of true tape-worms have been found in man, and two varieties of the *Bothriocephalus*. But two only of the true tape-worms are of frequent occurrence—namely, the *T. solium* and the *T. mediocanellata*. The former is the one endemic in this country; the latter is the more common tape-worm on the Continent and in South Africa.

The *Bothriocephalus latus* is endemic in some well-defined localities, chiefly in continental Europe; and the *B. cordatus* is new to science, having been but recently found in North Greenland (LEUCKART). These tape-worms have been known for a very long period; but they have not always been distinguished from each other. Indeed, the distinguishing characters are but recently known. They have often been confounded together under the name of "solitary worm," because it was believed they lived singly. This, however, is a mistake.

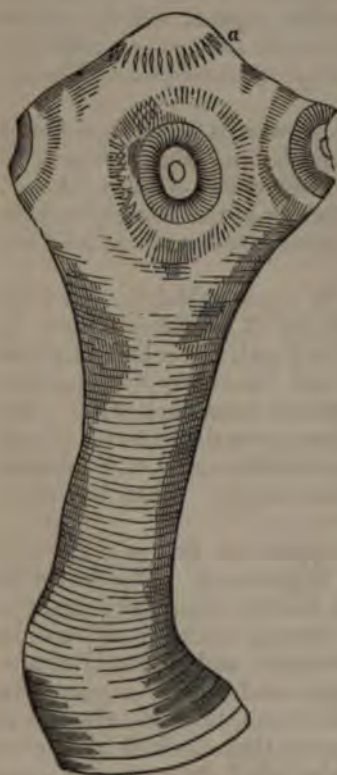
The *T. solium* and *T. mediocanellata* appear at first sight to be very similar to each other in general appearance. The latter is much the larger of the two. It is only in the alimentary canal—the small intestine of man—that they become sexually mature, in natives of France, Italy, Holland, Germany, and Great Britain. The *Tenia* has been also found in Egypt, and is very common in natives of Abyssinia;—so common is it there, that its absence is the exception to the rule. The affection is there looked upon as a natural occurrence; and so general is this belief, that when a slave is sold into Abyssinia he provides himself with a plentiful supply of kousso—the local remedy for expelling the parasite.

The Mature Tape-Worm Parasites.

1. *Tænia solium*.—It is the common tape-worm of this country, composed of segments of variable size, numbering from 800 to 1000; and these being endowed with considerable contractile power, the length of tape-worms varies greatly, and so also does the width and thickness. Nine to thirty-five feet may be quoted as average measurements of length. The body narrows from the posterior to the anterior extremity, till towards the head it becomes a mere thread. The parenchyma is soft and white, with microscopic calcareous particles, sometimes mistaken for ova (because they are round or oval), scattered through nearly every part.

The HEAD (Fig. 5) is very small, but it may be seen with the naked eye to be of a globose or triangular form, with black pigment ingrained into its substance, which may be the remains of blood. On the most anterior part of the head, with the aid of a lens magnifying twenty-three or twenty-five diameters, four circular projections, equidistant from each other, may be seen. Each has a

FIG. 5.*



circular disc or cup, surrounded by a rim of dense tissue. The parasite is able to elongate and retract these projections; so that, while opposite ones are put forward, the two others are kept back. Between the suckers, and anterior to them, is a convex protuberance or rudimentary proboscis, which is impervious, and surrounded by a double row of hooks (Fig. 5, a, also Fig. 6). These are siliceous, and number twelve to fifteen in each row. The shape of these is peculiar. They consist of a straight stem or handle, a middle knob, and a distinct hook or claw, surrounded by a sheath or sac. Bremser believes that a *tænia* loses these as it gets old; or it may shed them periodically by rows; and being lost, they may not be renewed, and so the parasite may be got rid of in the course of nature.

The head terminates a long and slender neck, on which there are transverse markings, but no visible joints or articulations. Such joints distinguish the body; and these joints, segments, or zoönites are united end to end in a single linear series.

The characters of the segments vary

* Head and neck of *Tænia solium*.—(a.) Circle of hooks.

at different parts of the body. They are square or oblong; and in the mature part of the animal the length of them is equal to twice the width. The anterior border of each segment unites with the anterior or previous segment, and is thinner than the posterior border, and is also narrower. The posterior border is thick, and projects or overlaps the border of the segment next in order, and is undulating or indented. The lateral margins incline to each other anteriorly. The two surfaces are flat or slightly elevated towards the centre.

Each mature segment contains male and female organs of generation. The opening at the side of each segment is the sexual aperture, indicated by a prominent papilla, once supposed to be a sucker. These openings are sometimes at one side and sometimes at the other. Two, three, or four consecutive segments may have them on the same side, or on opposite sides; but there is no regular alternation. With a lens a cup-shaped depression may be seen, showing two mesial apertures. From one of these a *lemniscus* or rudimental penis projects, connected with a horizontal (deferent) canal (sometimes indicated by dark pigmentary material) from a vesicular body in the middle of the posterior end of the segment (OWEN).

Behind this male orifice is the opening to the female organs, by a canal leading to a lobulated organ, which is the ovary or germ-stock. These parts are more distinctly developed the farther the segments examined are from the head end of the worm. While the head continues to adhere, by its circles of hooklets and oscula, to the mucous membrane of the intestine, the last or caudal joints, when they have arrived at sexual maturity, are separated one by one, or

FIG. 6.*

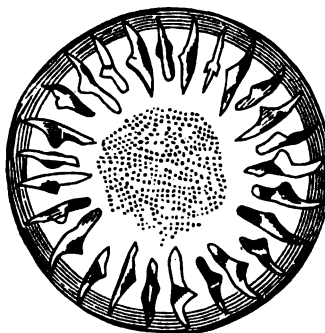
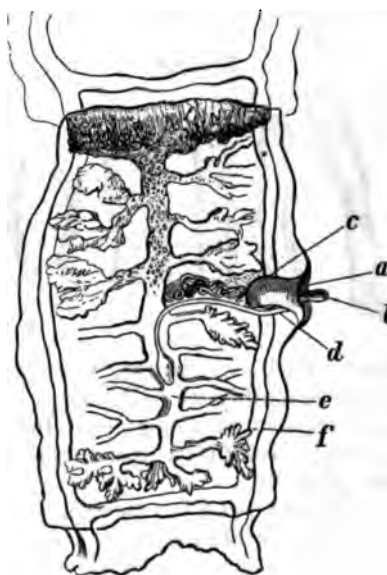


FIG. 7.†

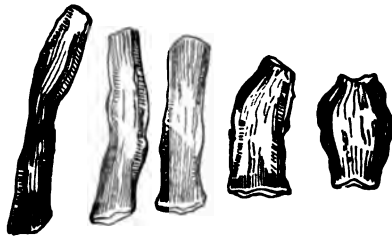


* Circle of hooks more highly magnified (after LEUCKART).

† Proglottis of *Tænia solium* magnified.—(a.) Genital pore, with its preputial cover or sheath-skin; (b.) Lemniscus or penis; (c.) The oviduct; (d.) The seed-vessel; (e.) The uterus; (f.) The water vascular system of vessels (after ROKITANSKY).

in numbers together, and new joints are at the same time gradually formed behind the head. Thus growth and development take place mainly towards the neck of the parasite, by a process of transverse fission; and thus a segmented individual or compound animal appears to grow. This segmentation of individual links by transverse fission ceases when the organs of generation begin to develop themselves in them; and when those are complete, the segment or link has arrived at sexual maturity or completeness. It is now called a *proglottis*. Thus all the new segments come to be developed between the head and those which are advancing to sexual completeness; and if the characters of complete sexual development be taken as the distinctive mark of individuality, then each segment of the tape-worm may be looked upon as a distinct animal; and this separation by fission or segmentation may be considered as analogous to what takes place in the medusæ or polypes—a kind of alternate generation, in which the *segments*, *zoönites*, or *proglottides* may be regarded as making up a colony of animals. It is only in the alimentary canal of man and other animals that the tape-worms, or cestoid entozoa, attain to sexual maturity; and in all of them the ova are fecundated before being discharged, and may often in the *T. solium* be perceived to have undergone the first stage of their development before they are excluded from the oviduct of the mature segment. The expulsion of the ova occurs in some one of the following ways:

FIG. 8.*



(1.) The impregnated segments separate from each other, and passing out of the body singly or in numbers with the fæces, or without any fecal evacuation, become decomposed, and so the eggs are set at liberty. The activity of these separate segments is retained for a considerable time after passing out of the body—a circumstance which led to their being at one time taken for a distinct

species of worm, to which the name of *Vermes cucurbitini* (from resemblance to a pumpkin-seed) was applied. The contracted appearances of a segment during its movements out of the body are represented by the forms shown in the accompanying woodcut (Fig. 8).

One may readily observe the activity displayed by these *beauties of nature* as they disport themselves on the recently extruded excrement of almost every constipated dog. The expelled joints may be seen to become violently contracted shortly after their expulsion, as if the stimulus of physical climate in their new situation provoked excessive contractions. The long single joints thus expelled become still more elongated by contractions of their transverse fibres, while the alternate contractions of these fibres with the

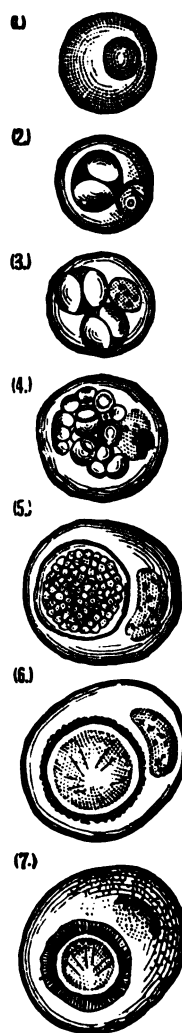
* Proglottides of a *Tænia (mediocanellata)* in various stages of contraction (after LEUCKART).

longitudinal ones cause shortening of the joint to such an extent that its breadth exceeds its length. Such a sequence of contractions produces movements which simulate those of progression in a worm, and thus these segments may be seen to move some little distance from the spot on which they may have first fallen, discharging ova during their march from the interior of the segment. (2.) Eggs are thus discharged through the genital pores of the mature segments; and if the segment be slightly squeezed, the ova may be pressed out. Such a contingency is not unlikely to happen within the rectum, when, by constipation or otherwise, the matured joints are retained, and constitutes one of the most serious dangers which the matured tape-worm inflicts on the animal it inhabits, and one of the strongest indications for its removal. It has been recently ascertained that in one or two instances the presence of a *Cysticercus cellulosæ* (the embryo of the *T. solium*) has been found to coexist with the previous prolonged existence of a *T. solium* in the intestinal canal of the human subject. (3.) The mature joints of the adult tape-worm seem, in some instances, to undergo a disintegration within the intestine of the animal they live in. Thus, Kuchenmeister on one occasion found the wall of the large intestine of a dog occupied by a white sandy powder, the particles of which, on examination under the microscope, turned out to be innumerable ova of a *T. serrata* which lived higher up the bowel, accompanied by its separated joints.

The mature segments are often expelled from the human rectum at the rate of six or eight a day, and they exhibit evidence of very active vitality for some time. Moisture is favorable for maintaining their existence, and for favoring the spread of the eggs over herbs, grass, ground fruit, or vegetables, which may become the food of man or of cattle.

The structure of these ova (Fig. 9) is peculiar; and the provisions possessed by their coverings for preserving the embryo are important points for consideration in connection with their transmissions through apparently impossible conditions into the bodies of animals, where they become further developed; and in connection with their powers of resistance to therapeutic agents (which have been called anthelmintics or vermifuges) administered for their removal.

FIG. 9.*



* Development of the ovum of *Tania solium*.—(1.) Previous to segmentation; (2, 3, 4, 5.) Segmentation in the impregnated ovum; (6.) Appearance of the early embryo, with its three pairs of siliceous spikelets; (7.) Mature condition of the ovum containing the embryo inclosed within its leathery case (after LEUCKART).

It is only in their earlier stages of development that they are really the analogues of ordinary ova. In the blind extremities of the oviducts of the mature joint of the tape-worm the shells of the ova appear to be composed of a calcareous transparent substance; and by the time the ova reach the central segments of the tube their hitherto transparent calcareous shell becomes not only much thickened, but is converted into a dark-yellow or brown mass, in the interior of which the embryo is formed, at first of the simplest structure, and most minute, being only about $\frac{1}{100}$ th part of an inch in size. The admixture of these organic elements with the calcareous shell imparts to it that extraordinary power of resistance to chemical, and even mechanical, violence which it certainly possesses. Dilute acids and alkalies have little immediate effect on this leathery husk; and even after hours of immersion in them, scarcely more than a slight swelling and transparency is produced upon the shell. It is, therefore, no matter of surprise that after months of exposure to warmth and moisture, or to cold and dry air, the pulpy, putrid, or dried-up mature segments of *tape-worms* should yield ova which show no sign of degeneration or decay.

A more or less speedy death of the expelled segments is followed by their putrefaction, hastened, it may be, by warmth and moisture. The eggs in their interior are then set free, to be carried by winds, waters, or other agents, wherever accident may determine. Thus they may lie to rot upon the soil, or they may be consumed as food by various animals which feed on such minute particles of food. The minority of these eggs, after many and long wanderings of this passive nature, may at length be engulfed unconsciously by some unfortunate animal with its food.

Within the alimentary canal of the animal which is thus so unfortunate as to eat the egg, a small embryo of most simple form is set free from the ovum by the rupture of the calcareous husk which incloses it. Such rupture is absolutely necessary to liberate the embryo, and may be effected by mechanical violence, such as friction, or crushing by the teeth in mastication of the food, rather than by solution or digestion in the stomach. Animal heat does not seem to be alone sufficient, nor is mere moisture sufficient to liberate the embryo; which when set free consists of little more than a highly contractile vesicle about the same size as the yolk of the ovum. It is peculiarly armed for progression by boring its way through the most delicate tissues. On one side of it are placed three pairs of spikelets; one pair points forwards, and the two other pairs are so placed that a pair is towards opposite sides of the embryo, or at right angles to the anterior pair. These spikelets are shown in the last two drawings of Fig. 9.

The *embryo* when free is named a "*proscotex*," and commences life on its own account by efforts at active migration. By the vigorous exercise of the spikelets it makes a passage through membranes, walls of vessels, and textures of solid viscera, so as to reach localities where it becomes encysted, and passes another phase of existence. The first portion of its path is pierced by bringing close together the several pairs of spikelets so as to form a kind of wedge-

shaped stiletto. The lateral pairs of these spikelets are then brought backwards to a rectangular position, and so they thrust the embryo forwards in the direction in which the anterior pair of spikelets pointed. Similarly repeated actions eventually accomplish progression to a resting-place; and the action may be aptly compared to the movements of the arms and attitude of the head of a swimmer. But this active migration is not the sole means by which the embryo *Tænia* is enabled to traverse the animal body. The embryo may penetrate a mesenteric vein, when it will at once be swept onwards by the current of the blood to the portal vein, and passing into the minute ramifications of the portal system, may find a resting-place in the liver. Leuckart has found the embryos of tape-worms in the blood in such large numbers that he inclines to regard the currents of blood in the vessels as the ordinary and more usual channels for the migration of the embryos. It also explains the wide diffusion of *tape-worm* embryos as *cysticerci* or *echinococci* in various stages of development throughout different viscera of the body, where they become encysted, and especially their very frequent site in the liver, peritoneum, and mesentery. Thus far completed and encysted, the embryo is called a "*scolex*." The embryos of *Echinococci* and *Cænuri* give rise to numerous *scolices*, which complete their development into tape-worms in the alimentary canal of another animal, when that animal happens to eat the liver or brain containing the cysts of such *Echinococci* or *Cænuri*; but the embryos of such *echinococci* or *cænuri* tape-worms find their way into man or animals with drinking-water, or with raw, uncooked articles of vegetable diet from moist soils, such as salads, roots, fallen fruit, all of which may be doubtless so exposed as to receive the germs or ova containing the embryos, passed along with fecal excrement of dogs especially, and which, after being dried, are carried by wind or water in all directions.

The third stage of development consists in the formation of segments, which are first seen in the form of marks, like girdles, surrounding that portion of the entozoön next to its oscula and hooklets, and which terminate in a caudal vesicle. It is now an incomplete segmented *Tænia*, and in scientific nomenclature is called a *Strobila*; and the development to this stage may occur while the entozoön is still within the closed cyst which has formed round it.

It is only in the alimentary canal of animals that the last and perfect stage of development is attained, by the tape-worm reaching sexual maturity. The segments or links marked off by the bands, joints, or girdles in the encysted *Strobila embryo* become mature segments by the development of sexual organs within them. This only takes place after the *Strobila embryo* has passed into the alimentary canal of an animal which can afford it a place to live and spend the rest of his days as a fixture attached by its hooks to the mucous membrane. The human alimentary canal is an oft-chosen place of the *T. solium* and *T. mediocanellata*. Here the tape-worm forms complete sexual segments or links, each being hermaphrodite, and tending to separate when completely mature.

After living for some time in this prolific condition, and having

produced often a very large number of joints and an enormous

FIG. 10.*



$\frac{1}{100}$ th of an inch \times 23 diameters.

quantity of ova, the existence of this troublesome parasite is terminated by the separation of the animal from its attachment to the intestinal membrane. When this separation occurs spontaneously, it may be that the circlet of hooklets being shed periodically, or being lost, they are not renewed, and so the prolonged life and romantic vicissitudes of a tape-worm may be thus brought to a natural termination. The whole length of the beast is then ignominiously expelled, while some reputed vermifuge, however innocent, may get the credit of its death. The apparent success of many such *parasiticides* is recorded and measured by yards of tapeworms, which, being ingeniously

bottled by worm-doctors and charlatans, are duly advertised to have been passed by John Smith or Sarah Brown, after they had been dosed with the "infallible" remedy.

The length of the tape-worm in the human body has been known to exceed thirty feet, and there are grounds for believing that the *T. solium* may attain to this size in the human intestine in about three or four months.

2. *Tania mediocanellata*.—This is a hookless, flat-headed tapeworm, the *cysticerci* and embryos of which are developed in the muscles and internal organs of cattle (LEUCKART); and man becomes infested with this tape-worm by eating imperfectly cooked veal and beef in which the *cysticerci* abound. It was first discovered by Kuchenmeister in 1855, and then shown by him to be different from the *T. solium*. Its head (Fig. 10) is large, obtuse, and truncated, and carries no hooks. Its sucking discs are much larger than those of the *T. solium*, as if to compensate for the want of the hooks. Its segments, when mature, separate easily. The ovaries are simple, giving off sixty lateral parallel branches. The eggs are similar to those already described.

The *T. mediocanellata* has been found in several instances of in-

* Head of the *Tania mediocanellata*, drawn with the camera lucida by Assistant-Surgeon B. J. Jazdowski from one of three specimens, all of which were removed from the small intestines of a soldier who died at Fort Pitt in 1860, and who had been for many years a cook at the Cape of Good Hope. The specimens are in the Museum of the Army Medical School at Netley.

valid soldiers who died at Fort Pitt, and at the Royal Victoria Hospital at Netley. In one instance three very large and long worms existed in the small intestines, each of them precisely similar in all respects. The soldier in whose intestine they were found died of *diabetes mellitus*, and he had been a cook for many years to a military mess at the Cape of Good Hope; another case was that of a soldier who had been also a long time at the Cape.

3. The *Tænia marginata*, produced from the *Cysticercus tenuicollis*, is only as yet known to infest man in its immature state as a *cysticercus*. The full-grown tape-worm being found in the dog and wolf, it is often confounded with the *T. serrata*, from which it differs in its comparatively bulky size and the peculiar form of its hooks. The proglottides nearly equal in size those of the *T. solium*. In its scolex or immature condition this parasite has a very wide distribution; for, in addition to its occasional presence in man, it has likewise been found in various monkeys, in cattle and sheep, reindeer, and in many other ruminants; in horses, swine, and even in squirrels. Its *habitat* is for the most part the peritoneum (ROSE and others). The *cysticerci* occasionally attain an enormous size.

4. The *Tænia elliptica*, whose *cysticercus*, or embryotic condition, is not yet known, is common to cats and dogs, and is known to infest man (ESCHRICHT, LEUCKART). Weinland believes that the *cysticercus* will be found in flies, and that dogs obtain the larvæ by snapping at dipterous insects.

5. *Tænia acanthotrias*, like the *T. marginata*, is only known in man as an embryo or *cysticercus*. From twelve to fifteen of them were found in the muscles of a woman about fifty years of age, by Dr. Jeffries Wyman in 1845. The woman was a subject in the dissecting-room at Richmond, United States. The rostellum of this parasite is furnished with three rows of hooks, fourteen in each row.

6. *Tænia nana*, when fully grown, attains a length of eight or ten lines, and carries from 150 to 170 joints. Its hooks are essentially the same in form as those of other *Tænia*, only they are very minute, and have a peculiar form, owing to the close approximation of the claw and of the anterior root-process (LEUCKART), which gives them a "bifid" appearance. Its head is comparatively large and obtuse, with a long neck. It was first described by Bilharz in 1851, having been found in Egypt in the intestine of a young man.

7. *Tænia flavopunctata* measures about eight to twelve inches long. The proglottides are short, and there is a yellowish spot, clearly visible to the naked eye, situated about the middle of each joint, which reminds one of the color and situation of the genital organs as seen in the *Bothriocephalus*. The reproductive orifices occur all along one side of the worm, and the eggs are unusually large. Only one instance of the occurrence of this parasite is on record: it was obtained in considerable numbers by Dr. Ezra Palmer in Massachusetts, in 1842, from an infant nineteen months old. They were expelled without medicine, their presence not having been suspected (WEINLAND, COBBOLD).

8. The *Tænia echinococcus* is very often seen in Iceland, where, in

its encysted immature state, it is the cause of a widespread endemic disease amongst the inhabitants. This *Tenia* is a very small one, consisting of not more than three or four segments, and not much longer than a line, and carrying twenty-eight to thirty-six hooklets. It is found in large numbers in the intestines of dogs.

The *Bothriocephali*, although classed with tape-worms, differ essentially from *Tenia*. Two species have been found in man, namely, —(1.) *Bothriocephalus latus*; and (2.) *Bothriocephalus cordatus*.

9. The *Bothriocephalus latus* is endemic chiefly in the north of Europe, and is found more especially in Russia, Sweden, Norway, Lapland, Finland, Poland, and Switzerland. The inhabitants of the French provinces adjoining Switzerland are infested with both species.

Instances of *Bothriocephalus latus* are said to have occurred both in England and France; but, when carefully inquired into as to their history, it will be found that this parasite maintains a very fixed geographical distribution. For example,—Of the six specimens in the College of Surgeons of England, one is from a native of Switzerland; one from a Russian belonging to the Russian embassy in London; one from a person who had been travelling in Switzerland; a fourth happened in the practice of Dr. Gull, in the person of a little girl from Woolwich, where there is always a number of foreign ships and sailors, bringing with them native food and water; another was passed by a native of Russia, who, after a long residence in England, paid a temporary visit to his birthplace, and returned to England with this parasite as a pleasant memento of his native country.

The liability to this form of parasitic disease appears to be greatest towards the sea-coasts and along river districts. Huss, of Sweden, describes it as extremely common on part of the Lapland frontiers, in Finland, and on the shores of the Gulf of Bothnia. On the extreme coast there is scarcely a family altogether free from it—old and young, rich and poor, native and emigrants, alike suffer from this worm; and in one or two large towns on the mouths of rivers at least two per cent. of the population experience its attacks. On passing inland the frequency of the disease diminishes, until, eight or ten leagues from the coast, rivers, or lakes, it almost ceases to be found. The natives believe it to be hereditary. Dr. Huss attributes it to the use of salmon (*Brit. and For. Med. Review*, l. c.).

FIG. 11.



The head of the *Bothriocephalus latus* is peculiar, and very different from the *T. solium*. It is of an elongated form (Fig. 11), compressed, with an anterior obtuse prominence into which the mouth opens; an opaque tract extending from the mouth separates two lateral transparent parts which are supposed to be depressions. There are no traces of joints till about three inches from the head; and throughout the entire body the segments have more length than breadth. The whole length of the mature parasite varies from six to twenty feet. It is of a grayish-white or yellow color; and the ova are very brown, giving the

mature segments a very marked appearance. The neck is not always obvious, for the worm has the power of making it long and thin or thick and short; and there are no joints or segments to be seen in it, but merely prominent ridges. The segments, when they become first apparent, are nearly square; but afterwards they become much wider than they are long. There are two orifices on one of the flat surfaces of each segment; the anterior orifice is connected with a male organ of generation, the posterior is connected with the female. The proglottides are never passed singly, but always in chains of many links, and particularly in February, March, October, and November. The ova (Fig. 12) are always discoverable in the fæces, of an ovoid form, with a perfectly translucent operculated capsule, through which the segmented yolk is distinctly visible; and at the period of discharge of the proglottides the ova show merely the stage of segmentation of the yolk. The *six-hooked* embryo, cased in a mantle studded with vibratory cilia, develops itself after segmentation, protected by the capsule in fresh water, for several months after the expulsion of the proglottides. When so far matured, the lid of the capsule opens up, and the ciliated embryo escapes (Fig. 12*a*), and becomes globular in shape, and moves actively about for a considerable period (a week). If during this period they do not succeed in obtaining access to the intestine of an animal adapted for their development, they lose the ciliated mantle, and perish. When these embryos are introduced by experiment into the intestines of mammals, the *scolices* and mature *Bothriocephalus* were found. Experiments in which living embryos were introduced by implantation between the brain and dura mater, and into the eyes of dogs, also under the skin of frogs, and by injection into the bloodvessels of mammals, give a negative result; *quoad* the development into *cysticerci* or *scolices*. So, also, feeding experiments with the *scolices* of the *Bothriocephalus* found in various fish lead to negative results; just as the feeding of fish with the eggs themselves. It is therefore justifiable to assume that drinking-water from lakes and rivers is the medium through which the living embryos of the *Bothriocephalus latus* find their way into the intestines of men and of mammals (Dr. J. Knoch, *Petersburger Medicinische Zeitschrift*, 1861; COBBOLD, *l. c.*).

FIG. 12.

FIG. 12*a*.

10. *Bothriocephalus cordatus*.—This species (Fig. 13, *a*) has only very recently been described by Leuckart, who received about twenty specimens from Godhaven, in North Greenland, one of which was from the human intestine. The parasite measures about a foot in length, and exists in dogs in considerable abundance. It differs from *Bothriocephalus latus* in the form of the head, which is heart-shaped (Fig. 13, *b* and *b'*), or obcordate, short, and broad, and set on to the body without the intervention of a long neck. The segments are distinct from the very commencement, near the head; and so rapidly do they increase in width, that the anterior end of the body becomes lancet-shaped. About fifty joints are immature; and in the largest example Leuckart counted a total of 660 joints. It dis-

plays a greater number of the calcareous corpuscles, and a greater number of lateral uterine processes (LEUCKART; Cobbold, "Remarks on the Human Entozoa," in *Proceedings of Zoological Society*, November, 1862).

FIG. 13.*



The Immature Tape-worms, Non-sexual, Cystic, or Vesicular Parasites.

These entozoa are variously spoken of by the older, and even by many recent writers on medical subjects, under the vague terms of *hydatids*, *cysts*, and *acephalocysts*. They all inhabit the closed cavities of animals, or they are inclosed in cysts in the more solid parenchyma of their organs. They are represented by the *scolices* or second stage of the *tape-worm* embryo, and consist of a *Tenia* head, provided with a similar circle of hooklets, and four oscula, and this head is united by a neck to a vesicular body of variable size. These are now believed to be, as already described, varied forms of *Tenia* embryos, of which the following are known to infest various parts of the human body:

1. The *Cysticercus cellulosæ*, as seen in man and in the pig, consists of a vesicle—conical, glistening, and white—containing fluid; to this a head is attached by a narrow pedicle or neck, which is transversely lined—the lines approaching to rugæ towards the vesicle. Its size varies in solid viscera from that of a small pea to a large marble; but in free cavities, such as in the ventricles of the brain, it attains a larger size. The head and neck can be drawn as if into the vesicle, so that the form and appearance of the parasite may thus be very much altered.

An external cyst incloses the parasite when it inhabits a solid viscus, such as the substance of the liver, or amongst the connective tissue of muscle; but in close cavities, such as the eye or the ventricles of the brain, there is no enveloping cyst, and the parasite floats free within the cavity. In these more free conditions it tends to grow more like the form of a *tape-worm*; and if it happens to be in the eye, it may soon destroy it, by fixing its hooks in some of its delicate textures. The cyst which envelops the parasite is developed at the expense of the tissue in which the parasite imbeds itself. They have been thus seen in

* (a.) *Bothriocephalus cordatus*, natural size; (b.) Head, back view, magnified five diameters; (b'.) Upper part of body and head, magnified two diameters.

the heart, liver, choroid plexus, the brain, in the tissue between the sclerotic and the conjunctiva, in the anterior and posterior chambers of the eye (MACKENZIE), and in the retina (GRAEFE). The head resembles that of the *T. solium*, and carries thirty-two hooks in two rows, and the neck varies greatly in length. The parasite is especially frequent in domestic swine, and in them it produces the appearance known as the "measles," or "measly pork."

2. The *Cysticercus ex tænia mediocanellata* is to be found in the muscles and internal organs of cattle. For our knowledge of the larval state of the *T. mediocanellata* we are mainly indebted to Professor Leuckart, of Giessen. He has artificially reared them in the flesh of calves, from the eggs of a *T. mediocanellata*; and recent experimental researches incontestably prove that the "measles" of cattle give rise to the *T. mediocanellata*. He fed two calves with the fresh eggs of the *T. mediocanellata*, by giving them the *proglottides* of this parasite. The first animal he experimented on died from a violent attack of the measles disease; and on dissection the muscles were found filled with measles, or vesicles containing imperfectly developed *scolices*. On the second occasion a smaller number of *proglottides* (in all about fifty) were administered, and the febrile symptoms again appeared with such virulence that Leuckart thought this animal would die also. Fortunately, after the lapse of a fortnight from the commencement of symptoms, some abatement of the disease took place, and this gradually continued until the animal was perfectly restored to health. Eight and forty days subsequent to the earliest feeding experiments (which were continued at intervals for eighteen days) Professor Leuckart extirpated the left cleido-mastoid muscle of the calf, and whilst performing the operation he had the satisfaction of seeing the cysticercus vesicles lodged within the muscles. They were larger and more opalescent than those of the *Cysticercus tænia cellulosa*, but nevertheless permitted the recognition of the young worms through their semi-transparent coverings. The heads of the contained *cysticerci* exhibited all the distinctive peculiarities presented by the head of the adult *Strobila* (the *T. mediocanellata*). Taking the results of this experiment in connection with previously ascertained facts, the most unequivocal evidence is brought together that man becomes infested with the *T. mediocanellata* by eating imperfectly cooked veal or beef in which the *cysticerci* abound.

3. The *Cysticercus tenuicollis* is rarely found in man, but it has occasionally been found in the mesentery and in the liver. Eschricht and Schleissner have shown that these *Cysticerci* are sometimes associated with the *Echinococcus* in Iceland (COBBOLD, l. c.).

4, 5, 6, and 7 require no special notice.

8. The *Echinococcus hominis* is the larva of the *T. echinococcus*; and the first accurate description of the immature form of the parasite was published by Bremser in 1821. These parasites have been and are still often indifferently named "hydatids" or "echinococcus cysts;" but English writers have restricted the term "*hydatid*" to designate the enveloping cyst, and the term "*echinococcus*" to signify the contained entozoön. The *Echinococcus* is an extremely common

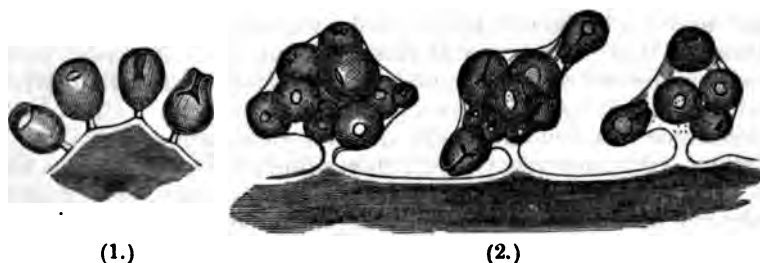
parasite of the human body. It has been found in the kidneys, lungs, liver, brain, heart, spleen, ovaries, breasts, tissue of the throat, and the bones; and they are not unfrequently discharged with the expectoration, or by stool. In the Icelandic endemic disease due to this parasite there is scarcely a part of the body in which it has not been found; and its occurrence in Iceland is a remarkable example of the prevalence of *cystic entozoa* in the human subject. It appears that the people of that country have been for some time suffering to a great extent under this very remarkable hydatid disease, which mainly affects the liver, peritoneum, and subcutaneous texture. Eschricht, writing to Von Siebold, says "the disease has extended itself to such an alarming degree that about a sixth of the whole population of Iceland are affected with it, and that it is attracting considerable attention at Copenhagen." It produces a long, protracted illness, terminating with a painful death, and means of cure have not yet been discovered. Von Siebold considers it "probable that this disease arises from the immense quantity of dogs kept in Iceland for the purpose of herding sheep and cattle" (Schleissner, *Medical Topography of Iceland*; ALLEN THOMSON, *l. c.*; also Leared, in *Medical Times*, 1863).

In some cases only a single "hydatid tumor" is developed in an organ or part; but occasionally two, three, or more tumors may be found. These "hydatid tumors" consist externally of a firm, fibrous capsule, of a tint which varies with the organ in which it may be developed. In the liver they are white, or of a yellowish tinge. The capsule adheres intimately to the surrounding tissue, and is abundantly supplied with bloodvessels. Bands of connective tissue may be seen stretching outwards from the capsule, and incorporating it with the tissue in which it is embedded. Within this capsule, and completely filling it, are—(1.) A gelatinous, translucent gray bladder or bladders, composed of numerous concentric hyaline layers, giving a laminated appearance to a section. It is finely granulated in some parts (degeneration?) and highly elastic. (2.) A very thin and delicate membrane is spread over the interior of this elastic hyaline bladder, as the innermost layer of the "hydatid tumor." This membrane is the mother sac of the *Echinococcus* embryo (HUXLEY), and corresponds with the germinal membrane of Professor Goodsir. It is studded with innumerable transparent cells, varying in extremes of measurement from $\frac{1}{100000}$ th to $\frac{1}{8000}$ th of an inch. It is the seat of the development of innumerable *Echinococci*; and to this membrane, in a fresh hydatid tumor, they are found connected by a delicate membrane, either singly or (more commonly) in clusters, the number of individuals on the cluster varying from ten to a hundred or more, as shown in the annexed wood-cut (Fig. 14).

On close examination with the naked eye these groups present the appearance of a number of delicate white particles upon the inner surface of the germinal membrane; and when the aggregation into groups consists of many individual embryos, they may be seen through a transparent cyst. They are the *scolices* or *embryos* of the *T. echinococcus* in various stages of development. The "hydatid tumor" is filled and distended with a clear watery fluid, with nu-

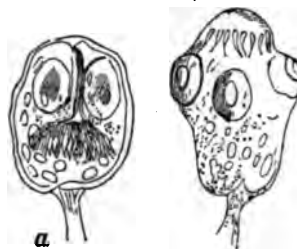
merous large and small vesicles, more or less clear and transparent, floating free, or so closely packed together that they cannot be removed without some degree of pressure. Some of them, particularly the smallest, adhere to the germinal membrane. They vary

FIG. 14.*



in size from that of a millet-seed to a size as large as a goose's egg, and their number not unfrequently amounts to several hundreds (560, PEMBERTON), or even thousands (7000 and 8000, ALLEN, quoted by PLOUCQUET and FRERICHs). The larger of these free vesicles sometimes contain smaller ones of a third generation, and occasionally they in their turn contain others of a fourth generation. The size of the "hydatid tumor" and the germinal membrane must increase and grow according to the number and size of the daughter vesicles, and in proportion to the quantity of contained fluid, which is sometimes rendered slightly opaque by the quantity of embryo *Echinococci* floating free in it. From the rotundity and distension of these inclosed vesicles it is difficult to fix them for examination; and when they are punctured, their fluid contents issue from the vesicle in a jet of considerable force, impelled by the contractile power of the elastic tissue; and if the incision be of a sufficient size, the vesicle will roll up, and turn itself inside out. The *Echinococcus* embryo varies in size from $\frac{1}{8}$ th to $\frac{1}{10}$ th of a line in length in the contracted state, and from $\frac{1}{10}$ th to $\frac{1}{8}$ th of a line in its elongated form. These variations in length are according as the head of the parasite is extruded or retracted within the vesicle. The contracted state, in which the head is retracted within the vesicle, is the form most commonly seen in the "hydatid tumor," after removal from the organ in which it was developed. In this state it is usually globular or oval, and slightly flattened at the opposite poles. In the elongated state, when the

FIG. 15.†



* Fig. 14.—Groups of *Echinococci*, showing—(1.) The pedunculated connection between these parasites and the germinal membrane; (2.) Their occurrence in groups, enveloped by a very delicately thin membrane, continuous with the germinal membrane (after ERASMUS WILSON).

† Fig. 15.—Two *Echinococci* from a "hydatid tumor." The one has the head retracted within the vesicle; the other has the head extruded.

head is extruded and the hooks appear outside, the parasite is usually larger at the cephalic end, where there may be seen four suckorial prominences and the circle of hooklets.

The *Echinococci* vary much in regard to the number of cephalic hooklets they display at certain intervals of growth, but not sufficiently to give ground for specific distinctions to be made amongst them; and while the number of hooks fluctuates in all the forms of *Echinococci* that have been described, the alleged differences in the size and character of these hooklets have reference to the degree of development of the parasite (LEUCKART, COBBOLD). These hooks are arranged in a double festoon, round a membranous disc (Fig. 16, A), and vary from twenty-eight, thirty-four, forty-six, or even fifty-two. They are arranged in two rows (Fig. 16, B), one row containing longer hooks than the other; the longer ones measuring about $\frac{1}{650}$ th of an inch. They each possess a gentle curve, so that there is a concave and a convex border, and a base (Fig. 17, b c) which

encroaches on the concave border for nearly half its length. The base is broader than any part of the hook, and has a bifid end. These hooklets move on the central bifid process as on a pivot (Fig. 17, f, g, k). Hook sacs may be distinguished with a sufficiently powerful lens. These hooklets are of so minute a size, and at the same time so extremely characteristic and important in diagnosis, that the annexed wood-cuts (Figs. 16 and 17, after ERASMUS WILSON) may give an idea of their shape and arrangement.

The head of the worm is separated from its body by a groove, and at its opposite pole is an umbilical depression, which gives insertion to the peduncle which fixed it to the germinal membrane (Fig. 15, a).

As the growth of the "hydatid tumor" advances, the external enveloping capsule gradually loses its uniform thin and smooth character. It becomes rigid, fibrous, or even cartilaginous, while its inner surface becomes rough and uneven, covered here and there with laminated deposits. Frerichs, of Berlin, has seen such capsules completely surrounded by a calcareous shell; and the thicker and more rigid the capsule becomes, the greater



* Fig. 16.—(A.) An *Echinococcus* viewed transversely, the head being directed towards the observer; s, s, suckorial discs. The hooklets are seen to encircle a membranous disc.

† Fig. 17.—(B.) The circle of hooklets seen upon its under surface; thirty-four in number, seventeen long and seventeen short. (C.) b, c, Lateral views of the separate hooklets—b, The base; c, The central extremity, or bifid process of the base; d, e, Hooklets viewed upon the concave or inferior border; f, g, h, A diagram illustrating the movements and position of the hooklets. The dotted line represents the outer surface of the neck, and runs through the fixed point of the three hooks.

is the resistance opposed to the further growth of the *Echinococci*, which may even be thus arrested, so that the parasites die, and a spontaneous cure results (CRUVEILHIER, FRERICHs.)

Compound "hydatid tumors" have been found in which the cavity is multilocular. Outgrowths or buds form, which give an alveolar character to the lesion (BUHL, VIRCHOW, FRERICHs.)

It has been usual to consider that there are two distinct forms of *Echinococci*, severally referable to different tape-worms; but they are now regarded as one and the same. The *Echinococci* so prevalent in Iceland are known to occur indifferently in men and oxen; and are sometimes so prevalent that about one-eighth of all the cases of disease are referable to this cause; and generally several members of one family suffer (LEARED).

Acephalocysts were first described by Laennec as growths of membranous cysts. In the present state of science they are regarded as abortive *Cysticerci* or *Echinococci*—parasites of one or other of those kinds in which the development has been arrested. Remains of hooklets have been found in them; and the structure of the cyst may sometimes be seen to be precisely similar to that of the *Echinococcus* cyst. They have been found in all those places where the *Cysticerci* and *Echinococcus* cysts are known to abound—*e. g.*, liver, spleen, kidney, bladder, and in the exostoses of bones.

Thus we have seen that each kind of *Tenia* has not only its own definite vesicular embryo, but each *Tenia* has a definite *Cysticercus* or *Echinococcus*; and all of them are capable of being developed or reared into *Tenia* when transferred into the alimentary canal of a suitable animal; and conversely, the development of *Cysticercus* and *Echinococci* occurs in man and other animals in consequence of segments of tape-worms, or the ripe ova they contain, being eaten with their food.

The experimental proof of these statements it is the principal scientific merit of Kuchenmeister to have established, by experiments which date so far back as 1851; and they have since been repeated and confirmed by many other observers (SIEBOLD, NELSON, ZENKER, LEUCKART, WEINLAND, KNOCH).

The experiments of Kuchenmeister were made on a variety of animals, and in one instance on the human body. The following is a short summary of his observations, together with those of Von Siebold and others, condensed from Dr. Allen Thomson's valuable paper in the *Glasgow Medical Journal*, No. x, July, 1855, and which demonstrates the

Relation between the Cystic and the Cestoid Entozoa.—These experiments mainly consist in observing the effects of feeding an animal with the ova or larvæ with which it is designed to affect it. The first experiment of this kind was performed by Kuchenmeister in 1851. He caused young dogs to eat with their food a number of the *Cysticercus pisiformis* of the rabbit and hare, and found that after some weeks these *Cysticerci* were converted, in the intestine of the dogs, into the *T. serrata*.

The more important of the experiments devised and performed

by Von Siebold confirm these results, and are described by him in his *Essay on Tape-worms*. They may be arranged as follows:

First Series.—Ten young dogs were fed with the *C. pisiformis* from the rabbit, and being killed and opened at different successive periods afterwards, the gradual progress of conversion of the *Cysticerci* into *Tæniæ* was carefully observed in their intestines. It appeared that, by the action of the gastric fluid in digestion, first the cyst and then the caudal vesicle of the *Cysticercus* were dissolved in the dog's stomach; but the head and neck, resisting entirely the solvent action, passed into the duodenum. Here they soon became attached to the mucous membrane; and after an interval of only two or three days they were seen to enlarge, the head and neck undergoing little change, but the body elongating and transverse grooves appearing, which afterwards became more marked, and divided the body into segments. In less than two months these *Tæniæ* had attained a length of ten and twelve inches; in three months they were from twenty to thirty inches long, and the reproductive organs were fully developed in the last or caudal segments, which then began to separate as *proglottides*.

Second Series.—These experiments were made by feeding young dogs with the *C. tenuicollis*, which is common in domestic cattle, and of which the vesicle often attains a large size. Having found that the vesicle was invariably destroyed by digestion, Von Siebold contented himself thereafter with giving the heads only to the dogs, removing artificially the vesicle. Six young dogs were the subjects of this experiment, which was conducted in a manner similar to the first, and with the same result, namely, the formation of tape-worms, which reached their full development in forty-eight days, and corresponded exactly with *T. serrata*.

Third Series.—In this set of experiments the *C. cellulosa*, from the flesh of the hog, was employed. Four young dogs received at different times a number of these *Cysticerci* with their food, and on being opened at different intervals afterwards, tape-worms, which resembled exactly the *T. serrata*, were found in their intestine, in various stages of advancement corresponding to the length of time that had elapsed. Von Siebold was struck with the close resemblance of this *T. serrata* of the dog to the common *T. solium* of man, and after an accurate comparison of various examples of these entozoa, concludes that they are identical, and not to be specifically distinguished, or that at most they are varieties of the same species, dependent only on the difference of their parasitic habitations.

Fourth Series.—This series of experiments was performed in the same manner as the last, but with the heads or scolices of the *Cænurus cerebialis*, the entozoön so well known in connection with the disease called "sturdy" and "staggers," which it produces when infesting the brain of sheep and cattle. In order that the *Cænurus* might be procured alive, the dogs experimented on were carried to a part of the country where a number of sheep were affected with the "sturdy." In the intestine of five out of seven dogs fed with the *Cænurus*, great numbers of *Tæniæ* were found, at successive periods and in different degrees of advancement; in

thirty-eight days the *Tæniæ* had arrived at maturity, and appeared, like those in the previous experiments, to correspond exactly with *T. serrata* and *T. solium*; in two other dogs the experiment was rendered nugatory by the dogs being ill of distemper at the time.

Fifth Series.—The last of the experiments related by Von Siebold were made with the *Echinococcus* animalcules of domestic cattle, which are probably not specifically different from those of man. As many as twelve young dogs, and also a fox, received a quantity of the small *Echinococci* in milk; and on being examined at various periods from the commencement up to twenty-six days, there were found, in all different stages of development, small *Tæniæ* totally different from any observed in the previous experiments, or indeed from any accurately distinguished or described by helminthologists. Von Siebold proposes to call this variety *T. echinococcus*. It is remarkable for its very small size—not much longer than an inch; and for the small number of its joints—which never amounted to more than three; and for the circumstance that the reproductive organs are confined to the two last segments, and the caudal joint separates as a *proglottis* at a very early period.

The last experiment to be noticed may be looked upon as the most interesting of all. Having the opportunity of repeating on a condemned criminal the experiments which he had previously performed on animals, Kuchenmeister contrived to give to this man, at seven successive times, between 130 and 12 hours previous to his execution, mingled with various articles of food, a number of *Cysticerci* from the hog and some from the rabbit. "They appear to have been partly disguised by their resemblance to the grains of rice in warm rice soup; partly by their likeness to the small bits of paste in a kind of vermicelli soup; and partly foisted on the unhappy wretch by being substituted for the small lumps of fat in blood-puddings" (*Brit. and For. Med-Chir. Review*, Jan., 1857, p. 119). After death, a number of young *Tæniæ*, in different stages of advancement, were found in the intestine; the greater number of them loose, but a few attached to the mucous membrane. The form of the hooklets, and other circumstances, induced him to regard these tape-worms as the *T. solium*. There were no traces of the *Cysticerci* last swallowed; and Kuchenmeister was of opinion that those only which were first taken, and which were quite fresh, had been converted into *Tæniæ*, and that those taken later, being dead, had been digested with the food. Leuckart made a similar experiment. He fed a man thirty years of age with *Cysticerci* from a pig, and in two months the man had *Tæniæ*.

But the enthusiastic Germans were not yet content with the proof. M. Humbert, of Geneva, experimented on himself. On the 11th of December, 1854, he swallowed fourteen fresh *Cysticerci* in presence of MM. Voget and Moulinié. Early in March of 1855 he felt the presence of *Tæniæ*, and discharged fragments of them.

With regard to the converse experiments, the following facts may be related:

Kuchenmeister, having previously caused the production of the *T. serrata* in a dog, by feeding him with the *C. cerebralis* from a sheep, gave to young lambs some of the ripe joints or proglottides of this *Tænia*, and by the fifteenth day the usual symptoms of *sturdy* began to appear in the lambs. Kuchenmeister sent some of the same *Tænia* to Van Beneden at Louvain, to Eschricht at Copenhagen, and to Leuckart at Giessen, all of whom, in separate experiments, caused lambs in the same manner to take the *Tænia* segments with their food, and in all the cases the same result followed—namely, the occurrence of the symptoms of *sturdy* at a period of from fifteen to eighteen days after the mature segments of *Tænia* were given. The same experiment had likewise been performed by Dr. Haubner, of Dresden. He caused six young lambs to swallow the living and mature segments of a *T. serrata*. They all died of the vertiginous disease; and the *Cœnurus* cysts were found in the brains, heart, lungs, and voluntary muscles. The symptoms commenced by the fifteenth day; and in point of time their appearance was curiously constant, the vertigo being obvious between the fifteenth and eighteenth days in all the experiments.

Several of these experimenters, having examined carefully the lambs so affected, were able to detect the progressive stages of formation of the *Cœnurus* in the cortical substance of the brain, where alone these entozoa seemed to attain the true *Cœnurus* form. There were abundant traces of them in the heart, diaphragm, and other muscles, and also under the skin, in some of the experiments; but in these situations they appeared to be abortive; while in the brain they gradually grew, and in some instances the vesicle had there attained the size of a hazel-nut. The brain was in all instances marked with inflamed grooves over its surface, indicating probably the track of the *Tænia* embryos; for at the end of each of these tracks, in the early stages, were found the minute *Cœnura*.

Another confirmation of the fact of the conversion of the ova of *Tænia* into *cystic entozoa* has been afforded by an experiment of Leuckart's, which merits separate mention. It gives the complement of the relation between the *C. fasciolaris* of the mouse and the *T. crassicollis* of the cat. Having in his possession a family of white mice, which he had employed for various experiments, and in none of which had the *Cysticercus* of the liver been perceived, he gave to six out of twelve, with their food and drink, the ova of the *T. crassicollis*, obtained by breaking up the ripe joints or proglottides of this tape-worm from a cat. Four months afterwards he found, on opening these mice, that four of them were affected with the *C. fasciolaris* of the liver; and he ascertained that in none of the mice which had not received the *Tænia* ova was there any production of these entozoa.

From the whole series of observations and experiments that have now been made, and of which a summary has been here given, the general conclusion may be drawn, that while much probably remains to be done in the details of the subject, a most important advance has through their means been made in the explanation of the manner in which these entozoa gain access to the seat of their para-

sitic habitations. It appears by them to have been ascertained—(1.) That entozoa are always introduced into animals from without ; (2.) That some obtain access to the body of animals from water, or other matters in which they have previously lived in the free condition, while others are taken along with animal food in which the entozoa have lived parasitically ; (3.) That entozoa, when reaching sexual perfection in their parasitic condition, require to be in a situation which communicates with the external air, their most common position being the alimentary canal, and more rarely the pulmonary cavities ; (4.) That almost all the entozoa inhabiting close cavities, or encysted in the bodies of animals, are only imperfect and earlier forms of other entozoa, which may attain maturity in the open cavities of the same or of different animals, or in the free condition ; (5.) That entozoa rarely propagate themselves in the same animal in which they have arrived at sexual maturity, but require a different habitation, which they reach by migrations in the various modes before referred to ; (6.) That the cystic entozoa are the imperfect states of different *Tæniæ* ; (7.) That *Tæniæ* are almost invariably introduced, in their earlier condition, into the bodies of animals with flesh or other animal food ; (8.) That if the ova of *Tæniæ* be introduced into the alimentary canal of a suitable animal, through water, vegetable food, or fruit, their tendency is, after penetrating the tissues, to become encysted, and to assume the form of a cystic entozoön, such as that of a *Cysticercus*, *Cænurus*, or *Echinococcus* ; (9.) That if these cystic entozoa again are taken by certain animals with their animal food, the head part (which corresponds with that of a *Tæniæ*) resists digestion, and has a tendency to establish itself, and become developed into some form of *Tæniæ* in the alimentary canal, by the formation of segments after attachment to the mucous membrane.

Many of the immature entozoa pass their whole life as encysted parasites, and a few even acquire the jointed form, or become partially divided into segments, while still within their closed cysts. A well-known example of this is afforded by the *C. fasciolaris*, which inhabits cysts in the liver of the rat and mouse, and has been the means of leading Von Siebold and Dr. Henry Nelson (independently of each other's observations) to the discovery of the remarkable relation now proved to exist between the *cystic* or *vesicular entozoa* and the *cestoidea* or *tape-worms*. These observers found the cystic entozoa in the liver of the mouse and rat in every stage of development, from the simplest vesicular form of the true *Cysticercus* to that which (from the number of the joints and their external form) has all the appearance of a true tape-worm, and from which, in fact, it only differs in the absence of sexual organs within the segments. A careful comparison of the form of the head, its circle of hooklets, the four oscula or suckers, and other parts in the *Cysticercus* of the rat or mouse, with those of the *T. crassicollis*, which inhabits the intestine of the cat, has shown an exact resemblance between them. Dr. Allen Thomson has repeated and confirmed these observations (Art. "Ovum," *Cyclopædia of Anatomy*). The conclusion such observations lead to is now generally regarded as established—namely, that the

cat receives its *T. crassicolis* with the flesh of the mouse or rat which it may have eaten.

Symptoms of the Presence of Tape-worm and Cystic Parasites.—From what has been stated, it is obvious that these must be very variable, determined by the form of the parasite and the locality in which it exists. Almost every case has some special symptoms of its own, local, reflex, or general.

In the alimentary canal the tape-worms occasion uneasiness, or pain in the abdomen, sometimes spasmodic, gnawing, or biting, but more frequently a distressing feeling which cannot be described. Irritation at the mucous orifices (mouth, nose, and anus) are also characteristic accompaniments. The bowels may be constipated or relaxed; the stools exceedingly dark or white; the appetite sometimes lost and sometimes voracious; sleep disturbed, and temper fretful; and often, as a result of so many combined irritating causes, a remittent fever occurs, which has received the name of "worm fever." The mind is also often so much depressed as to amount to hypochondriasis.

The symptoms of the existence of worms in the *small intestine*, such as *Tæniæ*, are often exceedingly obscure, and simulate many other diseases; so that, until a patient has passed a portion of the parasite worm, we are unable to predicate its existence with any certainty; and at no time till we actually see the parasite can we determine its species.

Prevention and Treatment of the Tape-worm and Vesicular Parasites.—The preceding history points out very clearly the means to be adopted for the prevention of *tape-worm*. At the same time, it is probable that there may be other accidental means by which the larvæ of the tape-worm may be introduced; and it will be easily understood how this may more particularly happen in the cases of butchers, cooks, or others in the habit of handling affected meat.

The instances in which the human body is affected with the *Cysticercus* or other *cystic entozoa*, though not very rare, are by no means so frequent as those of tape-worm; but they are much more serious in their effects, more obscure in their origin, and in the meantime, therefore, more difficult to prevent. Scarcely any attention has yet been given to the source from which the various individuals of the *cystic entozoa* infesting the human body may have derived their origin, but the observations already referred to make it extremely probable that the explanation of their introduction is to be sought for in the same causes which have been shown to operate in the lower animals. Thus it appears to have been demonstrated that the *Cœnurus* of the sheep proceeds from the ova or first embryos of *Tæniæ*, and it is most probable that those are obtained from the excreta of the dog. The only mode, therefore, of removing this affection from a flock in which it may have become prevalent, and in which it is well known sometimes to cause very great losses, must be the careful separation of the dogs from the sheep for a certain time; for such time, indeed, that the dog shall find no more *Cœnuri* in the offal of sheep which die of staggers, in eating which it receives the larvæ of its *Tenia*; and that the dog, being free from

this *Tænia*, shall not furnish, by his excreta, the ova or embryos which, being taken accidentally with the pasture or water by the sheep, establish themselves in them as *encysted Cænuri*. Von Siebold states the important fact, that those flocks which are entirely without dogs, and are stall-fed, are never affected with the "sturdy."

Prophylaxis, therefore, is all-important; and the entrance of the *scolices* must be prevented. The following remarks are not less revolting than suggestive. Dr. Gordon, of the Army Medical Department, thus writes:

"*Tænia* appears to be of very frequent occurrence among the white troops in Upper India, and especially the Punjaub; and I have been told by some medical officers who have been stationed at Peshawur, our nearest cantonment to Afghanistan, that they firmly believe every third soldier has had tape-worm during the two years regiments remain there.

"From what I have been able to ascertain on the subject, natives are not particularly liable to tape-worm, and certainly not more so in the northwestern parts of India than in Lower Bengal. This is generally attributed to their almost total abstinence from animal food; and when we consider that both Hindoos and Mussulmans—all except the very lowest classes—abhor pigs' flesh, while our own countrymen are very partial to it, and the common soldier probably not very particular regarding the early history of the animal that is converted into pork for his use, an additional circumstance in favor of the transformation of the *Cysticercus* constituting the 'measles' of pork into *Tænia* is thus disclosed to us.

"Those who have escaped the misfortune of having had to pass some years in India can form no idea of the vast herds of lean, half-starved pigs that roam over the fields and waste grounds in the vicinity of villages; neither can they have any conception of the nature of the food on which these pigs subsist.

"The natives of India perform their ordinary natural functions in the open air on a piece of waste ground left for the purpose on the outskirts of every village, and where, morning and evening, men, women, children, and pigs dot the ground at short intervals from each other. In an incredibly short space of time after the villagers have left the field it is as clean as if they had never been there, while the herd by which the clearance has been effected may be found in some shady place near or close to a tank, with the exception of a few of the more insatiable, that have gone to hunt for dead dogs, cats, cattle, and Hindoos that have paid the debt of nature since the previous meeting, and have been thrown or left on the plain to be devoured by domestic animals or vultures.

"Pigs, however, are not the only animals that live in this filthy manner in India; cattle and sheep, that are so particular in their food in Britain, acquire degenerate tastes in India; and it is needless to enter into similar particulars regarding ducks, fowls, turkeys, and pigeons, all of which are more or less used as food by our countrymen there.

"I have thus alluded to these matters with a view to indicate some circumstances that most unquestionably tend to vitiate the quality of the animal food upon which our troops in India must subsist, and I think I have at least shown a sufficient cause for almost any amount of disease in the bodies of these animals; as also why their flesh should be more liable to become diseased in Upper India than in Lower Bengal" (*Med. Times*, No. 357, May, 1857).

Abstinence from the practice of eating raw meat is to be strenu-

ously recommended; and cases closely inquired into will often be found to take their origin from the habit of taking animal food imperfectly cooked or *underdone*. Children have been affected with *Tænia* on being weaned, from the custom of giving them pieces of pig's flesh to suck in an uncooked state, and containing *Cysticerci*. Cooks and butchers are known to be more liable than other people to be infested with tape-worms; and in countries where raw or uncooked meat in the shape of fish, flesh, or fowl is much used, there tape-worms greatly abound, *e. g.*, Abyssinia. By contrast, this is remarkably brought out in that country. It is observed that those who abstain from flesh altogether in those countries are altogether free, *e. g.*, the Carthusian monks of Abyssinia.

Vegetables eaten green, such as salads, ought to be scrupulously clean, for it is through green vegetable food and fruits that the ova which lead to the growth of *Cysticerci* and *Echinococci* make their way into our bodies. A point of very great practical importance in preventing the spread of these encysted parasites is admirably insisted upon by Dr. Cobbold in the *Proceedings of the Linnæan Society*; and, based as it is upon a widely extended knowledge of the subject, it deserves serious attention. He suggests that in place of burying the excreta of animals known to be affected with *Tænia*, *all such excreta ought to be burned*. If they are simply buried, or merely allowed to drop and lie on the ground, multitudes of minute embryos escape destruction; and may ultimately find their way into the human body. *All entozoa (not preserved for scientific investigation and experiment), should be thoroughly destroyed by fire when practicable, and under no circumstances should they be thrown aside as harmless refuse.*

Medical Treatment.—For the ejection of *Tæniæ* from the small intestines a great many remedies have been recommended; but in the present day medical men very generally limit themselves to one or two methods, or to the action of a purgative. The celebrated Swiss remedy, purchased by the King of France, was a purgative composed of twelve grains of calomel and twelve grains of scammony, followed shortly after by half an ounce to an ounce of the sulphate of magnesia. In many cases it has been efficient.

By many the purgative treatment is seldom adopted, it being more usual to administer the *oleum terebinthinæ* alone. Half an ounce to two ounces of this medicine makes the patient slightly tipsy, is less likely to cause strangury, and produces three or four motions; and in these the worm is usually found, the animal having, it is supposed, a great sympathy to this substance, lets go its hold and actively attempts to escape. This medicine may be repeated twice a week. Three-fourths of the inhabitants of Cairo are said to be infested with *Tæniæ*, and the native remedy is twenty to thirty drops of *petroleum*—an agent not greatly dissimilar. The Grenadine bark has acquired much reputation in this disease in the West Indies, but it has not supported the hopes that have been entertained of it, at least in this country.

Drs. Christison, Jenner, Gull, and others, employ, with great success in tape-worm, the *ethereal oleo-resinous extract* of the male shield

fern (*Lastræa Filix-mas*) in doses of twenty to twenty-four grains—a remedy which by many is still believed to be the most efficacious. Dr. Gull's dose is one and a half to two drachms. The liquid extract of fern root is the officinal remedy of the British Pharmacopœia. It is made from the *rhizoma* or rootstalk of the *Aspidium* or *Nephrodium Filix-mas*, according to the formula: Fern root, in coarse powder, 1 part; ether, 2 parts, or a sufficiency: percolate and distil off the ether, and the liquid extract remains. The dose is ℞30 to ℞60, in milk, or with mucilage, and should be given on an empty stomach. Mr. Squire finds the extract of the unexpanded frond equally effective with that of the rhizome. The powder may be used alone in doses of one to three drachms.

A remedy sometimes used in Germany is Chabert's *bandwurmöl*, or the "*oleum Chaberti contra Tœniam*." It is obtained by the distillation of twelve ounces of oil of turpentine mixed with four ounces of the *oleum animale fetidum*, which is the crude oil obtained from hartshorn and animal bones.

A remedy of Abyssinian origin, called *Koussou* or *Cusso*, the flower of the *Brayera anthelmintica*, has recently been much recommended; and has doubtless been of great efficacy in some instances. It is administered in the form of powder, of which half an ounce is mixed with half a pint of warm water, and the infusion, *with the sediment*, is to be taken at two or three draughts, in the morning, on an empty stomach. If the bowels fail to be moved, a brisk cathartic ought to be given in three or four hours.

Another remedy, of more recent recommendation, is *Kameela* or *Reroo*, the *Rottlera tinctoria*, of the natural order *Euphorbiaceæ*, and sub-order *Crotoneæ*. It has been highly lauded by Dr. Gordon, Surgeon to the 10th Regiment of Foot. He writes,—

"The success and rapidity of effect of the kameela in removing tape-worm in the cases of soldiers of the 10th Regiment, to whom I administered it, were such that I did not consider it worth my while to keep notes of them after the first two or three; nor, indeed, were the men to whom it was administered latterly taken into hospital, for they soon became aware of the wonderful efficacy of the remedy, asking of their own accord for a dose of it, after which they invariably parted with the worm in the course of a few hours, and then went on with their military duty as if nothing had happened; while, as I afterwards ascertained, considerable numbers did not think of 'troubling the doctor at all,' but, on suffering from the characteristic symptoms of the worm, applied for the kameela to the apothecary, and always with the same effect.

"We prepare a spirituous tincture by adding Oj of alcohol to ℥iv of the powder, and then filtering. We never succeeded in obtaining more than ℥vj in this way; and of this ℥j in a little mint-water was generally found to be a sufficient dose, ℥ij being in some cases required, and perhaps in one or two, ℥iij, but I have never seen the remedy fail in removing the worm in a case where there were unequivocal symptoms of its presence.

"With kameela there is no unpleasant effect. It is not even necessary to take a dose of purging medicine as a preparative; and beyond a trifling amount of nausea and griping in some instances, no unpleasant effects are experienced; while by far the greater number of persons to whom it

is administered suffer no inconvenience whatever, beyond what they would from a dose of ordinary purging medicine" (*Med. Times*, 2d May, 1857).

One to three drachms of the powder, suspended in gruel, mucilage, treacle, or syrup, will of itself expel the worm.

[R. Rottleræ, ʒj; Oleoresinæ Filicis, fʒss.; Mucilag. Acaciæ, Syrupi Aurantii, aa fʒij. M. Sig. Take one-half at bedtime, and the remainder the next evening. It is advisable for the patient to fast ten or twelve hours before. Should it not operate adequately, a dose of castor oil should be given.—EDITOR.]

The duration of the parasite varies from a few months to thirty-five years (WAURUCH); and although it is considered by some absolutely necessary that the head of the animal should be expelled before the treatment can be considered successful, yet there are good grounds for believing that there is considerable chance of destroying the parasite if large masses near the head are brought away. The worm is then likely to perish; the most actively growing and important portions being discharged. The head of the worm ought always to be sought for during treatment; at the same time, not being easily found, the physician is not to be discouraged by not finding it. Out of 100 patients treated for *Tenia* by Bremser, he only once found the head in the dejections; yet all are said to have been cured.

FLUKE-LIKE PARASITES—*Trematoda*.

Definition.—Parasites of a flattened oval form, smooth, soft, and yellowish, or yellowish-brown, and not jointed or segmented. They are provided with two discs; one, situated at the pointed head extremity, forms a funnel-shaped depression leading to the oval orifice of a ramifying or bifurcating alimentary canal, which has no anal aperture. The other disc is situated on the abdomen, and terminates by a blind concavity. The opening of the sexual organs lies between the two discs. Male and female sexual organs are embodied in one and the same individual; and (as in parasites generally) they pervade a very large portion of the body of the mature adult animal.

Pathology.—A study of these parasites is of practical importance chiefly in relation to their ova and early embryotic forms, which are now known to be developed in the open waters, or in minute water animals. It is therefore incumbent on us to be able to recognize them, and to know something of their natural history. The generation of some of these *trematode* parasites constitutes one of the most remarkable parts of the history of the process of reproduction among the *Invertebrata*.

Two states or forms are known to exist—namely, a mature and an immature form—encysted and free.

It is only in the mature form that the generative organs are found. Such mature *Distomata* have their habitat in the biliary passages, especially of the *Ruminantia*, as well as in man. Among sheep the disease induced by *Trematodes* often commits great devastation, and is commonly known by the name of "the rot."

Self-impregnation and copulation are both possible in these parasites. It is only very recently that the facts connected with the natural history of these parasites have been ascertained, and their general result may be stated as follows: The fully grown and sexually mature *Trematodes* (as exemplified in the *Distomata*) are parasites of the higher *Vertebrata*, and are oviparous.

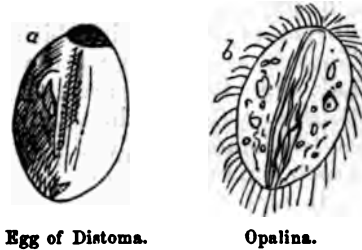
When the egg of the *Distoma* opens (by the springing open of a sort of hood, which gives it the operculated appearance at one end), it gives vent to an embryo which moves rapidly by means of cilia, as is the case with many infusoriæ, and especially of the *Opalina*, now regarded by Schultze, Agassiz, and Stein, as the earliest embryonic form of a *Distoma*. The discovery was made by Agassiz that a genuine *Opalina* (Fig. 18, *b*) was hatched from the egg (Fig. 18, *a*) of the *Distoma*. As such, they are found in sewage water (See A.

H. Hassall's *Reports on the Water of London*); also in the fæces of animals infested with liver flukes, their ova passing out with the bile. They die in pure water, as many vermicular animals do who would be more fortunate in water which is dirty, full of organic impurities, and abounding in food fitted for them.

From each of these ova is formed an embryo, in which no resemblance to the parent *Trematode* is to be recognized; but presenting the simple structure of a ciliated animalcule, like a polygastric infusorian known as the *Opalina*. This embryo is not itself converted by any direct process of development or metamorphosis into a perfect *Distoma*, but has a progeny gradually formed from germ-cells within it, and consisting sometimes of one, but more frequently of a number of bodies which, when they arrive at maturity, present each one an external form and internal structure and locomotive powers entitling them to be considered as independent animals. Nor are these directly converted into *Distomata*; a new progeny of animals is formed as before, nearly similar to those producing them, and equally differing from the complete *Distomata*. Each individual of this new progeny, as it increases in size, has formed within it, by development from germ-cells, the third progeny of the series and last of the cycle. These are different from their immediate parents, and in their internal organization soon manifest the type of the true *Trematoda*. They are endowed for a time with very active locomotive powers, to which a long caudal appendage contributes. Their progenitors have been confined in the parasitic condition, but these are in general freed from confinement, and move about with great vivacity for a time in the water surrounding the animals which their progenitors infested.

In this state they have been long known as *Cercariæ*, having the appearance of minute worms with tails, and were classed by

FIG. 18.

Egg of *Distoma*.*Opalina*.

Mueller, the Danish zoologist, with the *Infusories*. Regarding the history of the development of these animals, information is only fragmentary; yet so different periods have been observed in the development of different species of these *Trematodes*, that by analogy a tolerably connected history has been obtained of the whole process.

These *Cercariæ* in water are observed to be exceedingly lively and active, both as to body and tail; and after moving about free in the water they begin to go through a metamorphosis of a most extraordinary kind. They either become inclosed like a chrysalis in a pupa-case, or, penetrating the bodies of soft animals, or embryos of animals, inhabiting the water—*e. g.*, snails, fish, larvæ of insects, and the like—they become encysted within these bodies. They thus become parasites, for example, in the mussel family (*Mytilus edulis*); in the ovisacs of the *Gammarus*, or sandhopper (a small crustacean abounding on our sandy shores), and which may be seen leaping up in myriads from the beach; also in snails, frogs, newts, and the like.

In the cavities of these animals it lives as a ciliated animal, and multiplies by division of its body.

The next change in these parasites is that the cilia vanish. They fix themselves, and become by and by oval motionless bodies (STEENSTRUP, PRITCHARD, p. 270), which continue to grow, and in which a germinal mass becomes visible, of an elongated form, and out of which the first form of a *Cercaria* arises. It is a single-mouthed parasite, and is known as the *Monostoma mutabile*, inhabiting the intestinal cavity of water-birds.

In the interior of this parasite arises another embryo, which becomes free, and passes out of the bird into the water again; and being yet furnished with cilia, it makes its way especially into the aquatic larvæ of insects—*e. g.*, ephemera, caddice worms (*Phryganidæ*), the dragon-fly (*Libellulidæ*); and into the soft embryos of fishes like those of the cod kind; in snails, mussels, and such kinds of water animals, all used as bait and food for fishes.

Out of this ciliated embryo a *germ pouch*, as it has been called, makes its way; and from this germ pouch the *Cercariæ* broods are developed. In the eyes of fishes they inhabit the aqueous humors. Thus it seems probable that *Distomata* enter the human intestinal canal as *Cercariæ*, and pass thence into the biliary passages (FRERICHS); and according to the observations of Giesker and Frey, they may also penetrate directly into the skin, and undergo development in the subcutaneous cellular tissue. Two *Distomata* were found in the interior of a tumor on the sole of a woman's foot; and in all probability, as Frerichs remarks, the *Cercariæ* had entered the sole of this woman's foot while she was bathing in stagnant portions of the lake at Zurich. In another instance it was found in a cyst behind the ear of a sailor aged thirty-nine, and in an abscess in the head of a boy, twenty-five months old (Murchison, in Translation of Frerich's *Diseases of the Liver*, vol. ii, p. 488). The eating of uncooked fish, whelks, shell-fish, and uncooked garden snails, are

all obvious modes in which the *Cercariæ* of the *Trematoda* may find an entrance into the bodies of man and other animals.

Nine species of the fluke-like parasites have been found in man. They have been named as follows:

1. *Fusciola hepatica*, or *Distoma hepaticum*, in its full-grown condition, measures from eight to fourteen lines in length, and from three to six lines in breadth.

2. *Distoma crassum*.—In 1843 Mr. Busk found fourteen of these *Distoma* in the duodenum of a Lascar who died on board the "Dreadnought" hospital ship in the Thames. They are thicker and larger than those of the sheep, varying from one and a half to nearly three inches in length.

3. Of the *Distoma lanceolatum* only two instances are known of its occurrence in man.

4. Of *Distoma ophthalmobium* it is recorded that four specimens have been found in the eye of a child five months old, born with lenticular cataract. No one of them exceeded half a line in length; and they were situated between the lens and its capsule, where they could be recognized as so many dark spots on the surface of the lens (COBBOLD).

5. The *Distoma heterophyes* was found by Dr. Bilharz, of Cairo, in 1851, in the small intestine of a boy; and on a second occasion he collected several hundred specimens under very similar circumstances. The parts infested displayed a multitude of reddish points, due to the presence of dark-colored ova in the interior of the worms. The length of the largest specimens did not exceed three-fourths of a line (SIEBOLD, COBBOLD).

6. *Bilharzia hæmatobia* is so named by Dr. Cobbold in honor of its distinguished discoverer. It is, however, a bisexual parasite. The body of the male is thread-shaped, round, white, and flattened anteriorly. The oral sucker is triangular; the abdominal sucker at the end of the trunk is circular. Below this, at the curved margin of the abdomen, a furrowed canal exists for the reception of the female. This canal is peculiar and distinctive, and renders this *Distoma* generically distinct from the *Distomata* already noticed. The genital pore lies beyond the abdominal sucker and the commencement of the *canalis gynæcophorus*. The female is very thin and delicate; its tail is not provided with any canal. The suckers resemble those in the male; but the genital pore and the abdominal sucker are in contact. The length of the animal amounts to three or four lines, and the male is broader than the female (FRERICHS, MURCHISON, COBBOLD). Another name has therefore been given to it—namely, the *Gynæcophorus hæmatobius* (DIESING); but by whichever name it is known, it is of remarkable interest, not only from its peculiar anatomical structure, but from its great prevalence on the borders of the Nile, and from the grave and characteristic symptoms and appearances to which it gives rise. According to Griesinger it is met with in Egypt 177 times in 363 necropsies—i. e., equal to 33 per cent.

The first specimens were discovered by Bilharz, of Cairo, in the portal vein and its branches, and likewise in the walls of the urinary

bladder. It has been since observed by Griesinger, Reinhart, and Lautner in the veins of the mesentery, urinary bladder, ureters, and pelvis of the kidney, giving rise to a formidable and very prevalent disease. In the larger vessels, such as those of the liver, this *Distoma* gives rise to no derangements equal to those which result when it exists in the lining membrane of the urinary passages and the intestinal canal. There it induces hemorrhage and inflammation. In the intestine they are often associated with appearances resembling those of dysentery, with congestion, extravasation of blood, deposit upon and beneath the mucous membrane, fungoid excrescences, and croupy exudations that occupy ulcerated patches of the bowel. In many of these cases the eggs of the creature may be found wedged in long rows within the intestinal vessels, or in and beneath exudations on the free surface of the mucous membrane. Hence it was suspected whether the dysentery endemic to Egypt might not have to the presence of these *Distoma* the same relation as the itch has to the *Acarus*.

Such a conclusion receives a strong confirmation when we turn to the lesions produced in the urinary apparatus. Here the mucous membrane appears swollen in places which are covered with a soft, sandy, rotten mass, that is firmly fixed to the subjacent tissue. The microscope shows this mass to consist of the full and empty shells of the parasitic ova, embedded in a mixture of blood, exudation, modified epithelium, and crystals of uric acid. The thickening of the submucous tissue often produces stricture of the ureter, which is followed by retention of urine and all its dangerous consequences,—degeneration of the kidneys, pyelitis, dilatation of the pelvis, or atrophy of the renal substance; or the masses themselves become the nuclei of calculous deposits, and thus aid in the chlorotic exhaustion these creatures produce in the person they inhabit, by the consumption of blood they imply. Lastly, it seems not unlikely that the dislodgment of clots into the general circulation sometimes brings about pneumonia, in the way described by Virchow, and illustrated by the clinical researches of Kirkes (*Brit. and For. Med.-Chir. Review*, l. c., p. 125). Professor Virchow was good enough to show me a specimen, in his museum, of lesions in the bladder effected by the existence of this *Distoma*. The entire trunk of the portal vein is sometimes filled with this parasite, while their ova are found in the tissue of the liver; but the symptoms to which the *D. hæmatobium* gives rise are more referable to the urinary organs than to the liver. The urine is bloody, and sometimes contains the ova of the *Distoma*; and a state of profound cachexia supervenes (FRERICHS, COBBOLD, MOQUIN-TANDON). Dr. John Harley, of King's College, London, has recently directed the attention of the profession in this country to the remarkable prevalence of hæmaturia at the Cape of Good Hope—a condition which he found associated with the fertile ova of this entozoön passed in the urine. After micturition, a little blood, never exceeding a teaspoonful, or some dark coagula, like "veins," appear with the last half-ounce of urine. The urine itself is never bloody. Sometimes the blood-coagula will block up the urethra, and cause obstruction for a few minutes.

These are all the symptoms which appear in connection with the urinary apparatus; and numbers of people of both sexes are affected in precisely the same way in certain parts of the Cape—as endemic hæmaturia—especially at Uitenhage and Port Elizabeth. In various samples of urine sent to him by a person suffering from this affection, he invariably detected the ova of the entozoön. Of these he was kind enough to give me specimens, and a copy of the wood-cut (Fig. 19 A). He was successful in observing the perfect ciliated embryo after its escape from the shell.

Dr. Cobbold has discovered the same *Distoma* in the portal system of an African monkey.

7. The *Tetrascoma renale*, as its name implies, infests the tubes of the kidney, and was discovered in 1833 by Lucarelli and Della Chiaje. It attains a length of five lines, has an oval, flattened body, and is furnished with four suckers disposed in a quadrate manner at the caudal extremity. The reproductive organs are situated near the mouth.

8. *Hexathyridium pinguiicola* was once found in a diseased ovary. The parasite attains a length of eight lines (TREUTLER, OWEN).

9. *Hexathyridium venarum* has been found in venous blood, and from the sputa of persons suffering from hæmoptysis (TREUTLER, CHIAJE, FOLLINA). It attains a length of three lines, is cylindricolanceolate, with six suckers biserially disposed on the under side of the so-called head (COBBOLD).

Symptoms.—The symptoms to which *Distomata* give rise in the human subject must of course vary with the site of the parasite; but nothing definite is known regarding them, except in the cases of hæmaturia and dysentery, already noticed. In sheep their presence occasions dilatation and catarrh of the biliary passages, accompanied by atrophy of the hepatic tissue. Jaundice rarely shows itself, and then only lasts a short time; but ultimately a condition of anæmia is developed, under which numbers of sheep die. The disease is known as “the rot” among sheep; and it prevails to a considerable extent among flocks feeding on marshy and wet land near the shores of rivers.

In the human liver bodies have been found like the ova of entozoa, so frequently met with in the liver of rabbits (GUBLER, quoted by FRERICHs).

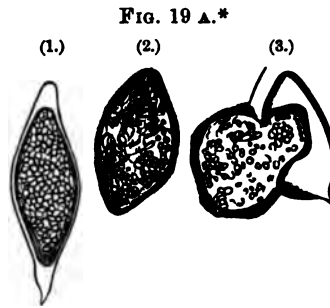


FIG. 19 A.*

* (1.) Ovum of *Distoma hæmatobium* from hæmaturia of the Cape of Good Hope (Dr. JOHN HARLEY); (2.) Embryo (ciliated) from ovum capsule; (3.) Embryo attached to the ovum capsule.

ROUND WORMS—*Nematelmia*.

Definition.—*Parasites of an attenuated and cylindrical form, having an intestinal canal suspended freely in a cavity of their bodies. They are possessed of a mouth and anus, and have the sexes distinct.*

Pathology.—In their mature state these worms inhabit the alimentary canal, the pulmonary tubes, or areolar tissue of man and animals. In their immature state some are encysted in the human body, and others come to maturity in the open waters. In the human subject they are represented by the *Ascarides*, the *Oxyurides*, the *Trichinæ*, the *Sclerostoma*, the *Strongylus*, the *Speroptera*, and the *Filaria*.

1. The *Ascaris lumbricoides*, or round worm, is perhaps the most anciently known, and is the most common of human entozoa. It is now regarded as specifically distinct from the *A. megalocephala* of the horse and the *A. suilla* of the hog (DUJARDIN, MOQUIN-TANDON, COBBOLD). It is much more common in children and adults than in old people. The body is long (six to sixteen inches), round, elastic, and attenuated towards both extremities, but more attenuated towards the anterior end. It is of a grayish red color, and sufficiently translucent to permit its viscera to be seen through its coverings. The *Ascarides* inhabit chiefly the small intestines, but may pass up into the gall-ducts, the stomach, the œsophagus, the nostrils, the mouth, or frontal sinuses; and there are cases on record and specimens in museums where the worm has evidently penetrated the coats of the intestine, and got into the peritoneum or into the pleura. It sometimes makes its way by the bile-ducts into the liver, and leads to hepatic abscesses. An interesting example of this may be seen in a preparation in the museum of the Army Medical Department at Netley. In some cases they are so numerous as absolutely to obstruct the intestines; in others only a solitary worm, or a pair may be found. Three distinct tubercles surround the mouth and characterize the genus. The posterior end is obtuse; but is straighter and thicker in the female than in the male. It is abruptly acute and curved in the male. An anus is situated in both sexes close to the tail, and in form is like a transverse fissure. In the female the body presents a constriction at the junction of the anterior with the middle third; and here the *vulva* is situated. The parasite throughout is marked with transverse furrows and with fine striae. Longitudinal equidistant lines run from the head to the tail, and are independent of the exterior envelope.

2. The *Ascaris mystax*, which infests every domestic cat, must now be regarded as a human parasite (PICKELLS, BELLINGHAM, COBBOLD). It varies from an inch and a half to two or three inches long, its head end being spear-shaped, in consequence of two lateral processes, from which it has been named *A. alata*. The ova have the embryo developed within them before they escape from the parent, and in this respect they resemble the *Oxyuris vermicularis*. (See description of the *Oxyuris vermicularis*.)

3. The *Trichocephalus dispar* occurs in the cæcum and colon, and was first noticed by Morgagni. A little more than a hundred years

ago (1760-61) a student of Gottingen was dissecting the valve of the colon of a girl five years of age. He accidentally opened the gut, and several of these entozoa came out. Wrisburg and other students considered the worm a new one; but the demonstrator of anatomy maintained that it was an *Ascaris* or an *Oxyuris*, and a dispute arose. At last the new parasite got a name as a new worm, and was called a *Trichinalis* or *hairtail*. But it afterwards turned out that its head was hair-like, and not its tail, so it has been since called *Trichocephalus*. About this same time an epidemic raged in the French army stationed at Gottingen, and was described under the name of the *Morbus mucosus*; and this entozoön was frequently found in the bodies of the soldiers who died of this disease.

It is said to be very common in persons attacked with typhus fever; and is found in those dying with excessive discharges from the bowels, as in cholera and diarrhœa.

It is found in France, England, Egypt, Ethiopia, and rarely in Italy; abounding particularly in the caput cœcum. It is generally thought to be scarce in England—a persuasion which Dr. Cobbold thinks has probably arisen from “the negligence of pathologists, whose arduous duties connected with the superintendence of post-mortem examinations have, perhaps, left them little time for these inquiries.” On the other side of the Channel this parasite is so abundant in some localities that not less than one-half the inhabitants of Paris are affected by it (DUVAINE).

These parasites are males and females, in separate sexes, varying from $1\frac{1}{2}$ to 2 inches.

The anterior extremity, carrying the head, is the narrow hair end, and it is usually buried in the mucous membrane of the intestines, while the remainder of the body moves freely in the midst of the mucous secretion, generally coiled upon itself.

The males are shorter than the females, and less thick posteriorly, with a long spiculum. The eggs are oval, with resisting shells $\frac{1}{10}$ th of an inch in length.

Generation of Round Worms.—The generative organs of these nematoid worms are adapted for the reproduction of an enormous number of fertile ova. They are male and female; but the males, as a rule, are scarcer and smaller than the females.

The fertility of these animals is enormous. Dr. Eschricht has made an elaborate calculation regarding the *A. lumbricoides*, the commonest parasite of man. The ova being arranged like flowers upon a stem in the ovary tubes, he has counted fifty in a circle, or in every transverse section. The thickness of each ovum he estimates $\frac{1}{800}$ th of a line ($= \frac{1}{800}$ th of $\frac{1}{2}$ th $= \frac{1}{1600}$ th of an inch); so that in every line of length of the worm there would be 500 wreaths of 50 eggs each, = 25,000 eggs.

The length of each horn of the uterus is taken at sixteen feet, which gives 2304 lines; and for the two horns it will give 4608 lines. The eggs, however, gradually get as large as $\frac{1}{80}$ th of a line, so that only sixty wreaths of eggs come to be on one line, or about 8000 ova; and an average gives 14,000 ova in a line—i. e., sixty-four millions of ova in every mature female *Ascaris*.

What becomes of all these ova? The embryo is not developed within the body of all of these parasites; and the source of the various *Ascarides* which inhabit man is not yet fully known.

The ova being discharged by millions, many of them in large cities will be carried into streams of water. An extremely small proportion is ever likely again to find their way into the alimentary canal of the animal which was the dwelling-place of the parents of these ova. Thus they become food for numerous minute inhabitants of the water, and therefore stand in the same relation to many of these animals as the *cerealia* in the vegetable kingdom to the higher animals. Indirectly they thus contribute to the sustenance of man and animals.

But on the other hand there are many circumstances which tend to show that the *A. lumbricoides* is most frequently introduced as a minute embryo with water or with fresh uncooked vegetable food.

Dr. Paterson, an eminent physician in Leith, observed that certain families who drew water from a public well in a particular street there, were very subject to the *A. lumbricoides*; while towards the other end of the same street the families were supplied by the pure water which supplies Edinburgh and its vicinity; and these families were free from the parasite. The water of the well came from a dirty pond or lake in the vicinity (called Lochend), and in its water numerous vermiform animalculæ existed, such as the *Amphicæcia fluviatilis*, perhaps the embryonic form of an *Ascaris*.

Another point to be remembered in all inquiries of this kind is the intense tenacity of life, and revival from a state of apparent death exhibited by these parasites; and in no class of animals has the origin by spontaneous generation been more strenuously contended for than in regard to these entozoa. For example, there is a minute worm of a nematoid kind which is a parasite upon wheat grains (the *Vibrio tritici*); some of these being dried, and then re-moistened after a lapse of four to seven days, they resumed their living active state (Bauer in *Phil. Trans.*, 1823, p. 1, quoted by Owen). Dr. Blainville has given other similar instances of revival after desiccation; and mature entozoa will even resist the effects of such destructive agents as extremes of heat and cold, to a degree beyond the endurance of any other minute animals. Owen relates that a nematoid worm has been seen to exhibit strong contortions—evident vital movements—after having been subjected above an hour to the temperature of boiling water with a codfish which it infested. Rudolphi mentions of some entozoa which infest herrings annually sent to Berlin hard frozen and packed in ice, that they do, when thawed, exhibit unequivocal signs of restored vitality (Owen, *Lectures on the Invertebrata*, p. 80). *Ligulæ* are often found alive in undercooked codfish. Rudolphi found individuals of *Ascarides* (*spiculigera*) stiff and hard in the gullet and stomach of a bird (*cor-morant*) kept in spirits of wine for eleven days, and which returned to life in warm water. Miram has seen individuals of the *Ascarides* (*acus*) from the pike become dry, and remain sticking to a board, where they would revive again by being placed in water, and in some instances they would move a part of the body which had im-

bibed the fluid, while the remaining part continued shrivelled up, and adherent immovably to the board. I have seen the very same results in the *Ascaris* which infests the peritoneal covering of the mackerel. Such being the tenacity of life on the part of the mature animal, how much more do the ova possess the powers of endurance? Without losing latent life, they even develop themselves under circumstances of the most improbable kind. Dr. Henry Nelson and Dr. Allen Thomson have observed the development of the ova of the *A. mystax* to proceed for several days, while the parent bodies containing them were immersed in oil of turpentine. I have once seen the same occurrence; and also I have seen the development of the embryo proceed in spirits of wine for about three weeks before signs of vitality had ceased.

Periods of Incubation of the Ova.—The eggs are ovoid, and covered by a transparent envelope or chorion, which after fecundation and segmentation becomes tuberculated. Hence the various accounts given as to their surface appearance. They are expelled with the faeces in the case of the *A. lumbricoides*. They have been placed in water and taken care of for various lengths of time, and Richter records that at the end of eleven months each ovum contained a living embryo. In August, 1853, Verloren and Richter put a fragment of a mature female *Ascaris* (*marginata* of the dog) into water, so as to keep the ova moist merely; and he examined them from time to time with the microscope. Segmentation having commenced, the development of the young was completed in fourteen days. They moved with great briskness within the egg-shells, *but did not break through them*. In this imprisoned or encysted state they continued throughout autumn and winter; the movements of the embryos gradually diminishing and at last entirely ceasing during the winter months, to recommence in the following spring, and become again distinct in summer; *but they never broke through the shell*.

The condition of these *Ascarides* from the encysted state of inclosure within the egg is only changed under favorable circumstances—namely, when the animals are liberated and carried on to further development; and now it is known that the embryo of nematoid worms may pass the winter in a torpid state, floating about in the open waters, or lying in moist places. The fully formed embryo is cylindrical, its length $\frac{1}{10}$ th of an inch; the mouth is not furnished with the three characteristic papillæ of the genus, and the tail terminates suddenly in a point. It is highly probable, from the evidence, that the embryos are directly transferred to the alimentary canal of man from river- and pond-water.

4. The *Oxyurus vermicularis* was known to Hippocrates, and is one of the most troublesome parasites of children, and occasionally of adults. It is a minute, white, thread-like worm, the male being about a line and a half in length, and the female five or six lines. They inhabit chiefly the rectum, where they are often found in clusters, rolled up in balls of considerable size, and from the rectum may creep into the vagina or urethral orifice. Sometimes they give rise to profuse and exhausting bloody discharges from the vagina.

The eggs of this parasite have embryos developed within them prior to their escape from the parent; and in this respect they differ from the *A. lumbricoides* and the *Trichocephalus* on the one hand, and from the viviparous *Dracunculus* on the other. In this character, however, they resemble the *A. mystax*.

In all probability the young escape from the eggs soon after the latter are expelled or migrate *per rectum*, and, like others of the *Nematelmia*, gain access to the human body with our vegetable food or water whilst still in a sexually immature condition.

5. The *Trichina spiralis* [an animal which gains access to the human body through the eating of the flesh of animals infested with it], was first seen by Tiedemann in 1822, and was first described by Mr. Owen in 1835 from a specimen taken to him by Mr. Paget. It has since been often recognized chiefly as a dissecting-room curiosity. The student tries in vain to clean the fibres of the muscle he is dissecting, which, however clearly displayed, still looks as if it were "sprinkled over with the eggs of some insect."

Instances of the *T. spiralis* in the muscles of the human body are of much more frequent occurrence than has generally been supposed. Most probably, from their very minute size, they have hitherto been overlooked. Virchow had not seen a case before 1859, after which he met with no less than six in one year. It is perhaps more common in man than even the *Cysticercus*. Zenker, of Dresden, found *Trichinae* in four out of 136 dissections—i. e., one in thirty-four.

Of late, however, much interest has been excited in this subject; for, in place of the *T. spiralis* being quite a harmless parasite, as has been hitherto the belief, Zenker has lately shown that it is the source of a new and most alarming form of disease;* that, in place of remaining harmless and encysted in its capsule, only to cretify or degenerate, it may free itself from this cyst, and, migrating amongst the muscles, may give rise to symptoms of the most serious kind, causing death in a strong and healthy person after a few weeks of painful suffering.

It seems, indeed, to be the most dangerous of all parasites—not even excepting the *Echinococcus*; and it behooves the physician to know something about the nature, origin, and development of this entozoön, seeing that its pathological relations are now known to be of extreme importance and interest.

The *T. spiralis* has been hitherto known as a minute round worm, inclosed in a more or less transparent capsule, lying between the sarcolemma of the primitive muscular fibres (Figs. 20, 21); but when the parasite is free, it finds its way within that sheath. It forms the type of a distinct genus of *nematodes*, having no genetic relations with the *Trichocephalus dispar*, as was supposed; but is reproduced viviparously.

[(1.) The *Trichina spiralis*, in its mature state, is an extremely minute

* [Although the trichinous disease, in respect to its symptomatology and danger to life, has been known only since 1860, there is now no doubt, that it has occasionally happened in Germany for a long period, and was confounded with other diseases, particularly typhoid fever.]

nematode helminth; the male in its fully developed and sexually mature condition, measuring only $\frac{1}{8}$ th of an inch, whilst the perfectly developed female reaches a length of about $\frac{1}{4}$ " ; body rounded and filiform ; usually slightly bent upon itself, rather thicker behind than in front, especially in the males ; head narrow, finely pointed, unarmed, with a simple central minute oral aperture ; posterior extremity of the male furnished with a bilobed caudal appendage, the cloacal or anal aperture being situated between these divergent appendages ; penis consisting of a single spicula, cleft above, so as to assume a V-shaped outline ; female shorter than the male, bluntly rounded posteriorly, with the genital outlet placed far forward, at about the end of the first fifth of the long diameter of the body ; eggs measuring $\frac{1}{1270}$ " from pole to pole ; mode of reproduction viviparous. (2.) The sexually mature trichina inhabits the intestinal canal of numerous warm-blooded animals, especially mammalia (also of man), and constantly in great numbers. (3.) At the second day after their introduction, the intestinal trichinæ attain their full sexual maturity, lose their spiral figure and become stretched, whilst they grow rapidly, and their generative organs are developed. (4.) Most females contain from three to five hundred ova. In six days the female parasites will contain perfectly developed and free embryos in the interior, and these on attaining full size pass out at the vaginal opening. The eggs of the female trichina are developed within the uterus of the mother, into minute filaria-like embryos, which from their sixth day are born without their egg-shells. (5.) The new-born young soon afterwards commence their wandering. They penetrate the walls of the intestines, and pass directly through the abdominal cavity into the muscles of their bearers, where, if the conditions are otherwise favorable, they are developed into the form hitherto known. (6.) The direction in which they proceed is in the course of the intermuscular connective tissue. (7.) The majority of the wandering embryos remain in those sheathed muscular groups which are nearest to the cavity of the body (abdomen and thorax), especially in those which are smaller, and most supplied with connective tissue. (8.) The embryos penetrate into the interior of the separate muscular bundles, and here already after fourteen days, acquire the size and organization of the well-known *trichina spiralis*. (9.) Soon after the intrusion of the parasite, the infested muscular fibre loses its original structure. The fibrillæ collapse into a finely granular substance, whilst the muscular corpuscles change into oval nucleated cells. (10.) The infested muscular bundle retains its original sheathing up to the time of the complete development of the young trichinæ, but afterwards its sarcolemma thickens and begins to shrivel at the extremities. (11.) The spot inhabited by the rolled-up parasites is converted into a spindle-shaped widening, and within this space, under the thickened sarcolemma, the formation of the well-known lemon-shaped or globular cysts commences by a peripheric hardening and calcification. One cyst may have from one to three trichinæ. (12.) The migration and development of the embryos also take place after the transportation of impregnated trichinæ into the intestines of a new host. (13.) The further development of the muscle trichinæ into sexually mature animals is altogether independent of the formation of the calcareous shell, and occurs as soon as the former have reached their completion. The male and female individuals are already recognizable in their larval state (LEUCKART, COBBOLD*.)]

* [Entozoa: an Introduction to the Study of Helminthology, with reference more particularly to the Internal Parasites of Man. By I. Spencer Cobbold, M.D., F.R.S. London, 1864.]

The non-encysted *Trichina* may exist in the flesh of animals without being visible to the naked eye. In the encysted state they are difficult of detection without the aid of a lens, if cretification has not commenced in the cyst. The cysts are round or elongated, and appear, according to their shape, like small round dots, granules,

FIG. 20.*



FIG. 21.†



100th of an inch X 300.

vesiculæ, or streaks, grayish-white or opaque, and quite distinct from the red transparent muscle. When the cyst has become calcareous, its limy material may be dissolved away by acetic or hydrochloric acid with the evolution of gas, and the parasite is then seen coiled up within (Fig. 20).

The symptoms of the disease induced by this parasite are at first of a febrile nature, having a close resemblance to some forms of specific fevers. Dr. Philip Frank, lately Assistant-Surgeon on the staff of Her Majesty's Army Medical Department, was the first to send an account of this remarkable disease from Germany to this country. He described a case of its occurrence in the *Medical Times and Gazette* of May 26, 1860; and recently Dr. Parkes has given a short notice of *trichina disease* in the *Sanitary Report of the Army Medical Department* for 1860, p. 351. The history of the case referred to by Dr. Frank is as follows:

In January, 1860, a servant girl about twenty years of age died in the Dresden Hospital from the effects of the *T. spiralis*; and the muscles of her body furnished materials for numerous observations and experiments, which have thrown much light on the origin and development of this parasite. The illness of the patient commenced about Christmas, 1859; and the symptoms may be arranged into two sets,—(1.) Extreme lassitude, depression, sleeplessness, loss of appetite, and eventually febrile phenomena, which were well expressed, so much so, that the case was set down as one of typhoid fever; but grave doubts prevailed, for (2.) A new train of symptoms developed

* Slightly magnified cyst of *Trichina spiralis* (after VIRCHOW).

† The *Trichina spiralis* removed from its cyst (after VIRCHOW).

themselves—namely, the whole muscular system became the seat of excruciating pain, especially in the extremities. Contractions of the knee and elbow joints supervened, and associated with such extreme pain as to render extension of the limbs impossible. (Edema of the legs followed; and the case terminated fatally by pneumonia, about the twenty-eighth or thirtieth day after the first symptoms of illness.

A post-mortem examination of the body showed the muscles moderately developed, of a pale reddish-gray color, and dotted over with specks, which turned out to be groups of non-encapsuled *Trichinæ*, lying free upon and within the sheaths of the muscular fibres. They were alive—some coiled up and others lying straight; and they appeared to be in all stages of development, diffused throughout all the striated muscles of the body, not even excepting the heart itself. They abounded in such vast numbers that as many as twenty *Trichinæ* were seen in the field of view through a low magnifying power, the muscular tissue being everywhere in a degree of very marked degeneration. In the jejunum were found sexually mature *Trichinæ*. Death was due to the development of the *T. spiralis*, whose existence fully explained the anomalous symptoms which attended the case.

On looking into the history of the girl it was found out that she had been a servant in a farm-house, and had been taken ill very soon after the killing of two pigs and an ox—animals which it is customary to kill about Christmas.

Pigs are known to be infested with the *T. spiralis*—so are oxen; and Professor Zenker went to the master's house, and found some ham left of the identical pig that had been there killed, and also some sausages. The flesh of the pig was examined microscopically, and every specimen examined showed that the pig's flesh was infested with *Trichinæ* in the encysted state. At the same time Professor Zenker learned that, soon after the girl had been taken ill, the housekeeper became unwell, with similar symptoms, but in a less severe degree; and all the servants about the farm became more or less ill about the same time. The house of the butcher who had killed the pig was then visited by Professor Zenker, who was informed by the wife of the butcher that he had been very ill since that event. He had been three weeks in bed, suffering from *rheumatic pains in the limbs*, and had been as if paralyzed over his body—unable to move his arms, legs, or neck. He had never suffered anything of the kind before, but had always been a healthy and strong man. He thought he had caught cold the day he killed the pig; but when it is known to be a habit of German butchers to taste the meat they kill, in the raw condition, the history of these cases, to Professor Zenker, became a history of *trichinatus disease*—the development and growth to maturity of the *T. spiralis* in the muscles of those who lived at the farm-house, as well as of the butcher who had killed the pig, and who no doubt had eaten some of its flesh.

Numerous experiments were made with the flesh of the girl who died in this remarkably morbid state. Portions of the flesh were

sent by Zenker to Professor Virchow at Berlin. He fed a rabbit with some of it, and this rabbit died about a month after the feeding, with symptoms of general muscular paralysis, and myriads of young *Trichinæ* were seen in its muscles. Other rabbits were fed with the flesh of the first rabbit, and they too died with similar phenomena.

Another observer had before made similar experiments. Herbst, in 1852, fed three young dogs with the flesh of a badger whose muscles were saturated with *Trichinæ*. The dogs in their turn became *trichinatus*; being killed after a few months, the parasites were seen in their flesh. Pigeons also were fed with moles' flesh known to be *trichinatus*; and free *Trichinæ* were found in the flesh of the neck, the wings, and the thighs of the pigeons in eighteen days. But Herbst did not examine into the relation between the encapsuled and the free *Trichinæ*, as Virchow and Zenker have done.

The *Trichina spiralis* is now well known not to be limited to the muscles of man. It occurs in eels, cats, dogs, badgers, hedge-hogs, pigeons, moles, and swine.

Virchow found the villi of the intestines of the rabbits loaded with the ova or *prospermiæ* of the entozoa; and he found mature *Trichinæ* of both sexes moving freely in the mucus of the intestine. The males were filled with sperm corpuscles, while the females were densely stocked with ova and their germs, and with young ones in the eggs, coiled up like little snakes.

[It has been found also in the horse, ox, sheep, and other ruminants, also in rabbits, rats, mice, and guinea-pigs, which have been fed with trichinous flesh. The debris of an animal devoured by carnivora may become fatal to rodents, or a carcass near a marsh or rivulet may communicate the parasites to the ruminants which drink the water, or to pigs (DAVAINE).]

In the summer of 1860 a subject was received into the dissecting-room of the University of Edinburgh; and the muscles of that subject contained numerous flesh-worms. Dr. Turner, the demonstrator of anatomy, then took the opportunity of verifying the experiments of the German professors. He fed kittens with portions of the human flesh containing the worms, which were observed to move, though somewhat languidly, on rupturing the cysts. To one cat on the 7th, 13th, and 16th of July he gave portions of the flesh, and in the intervals fed it on bread, milk, and fish. He killed the cat on the 24th of the month. Nothing could be seen with the naked eye in the fluid of the small intestines; but on placing a drop below the microscope, thread-like worms were seen actively moving about in it, or coiling themselves up in a spiral form. Every drop of fluid taken contained one or more. Each of the thread-like worms was about $\frac{1}{10}$ th of an inch long and $\frac{1}{1000}$ th of an inch broad, with a pointed and rounded end, and about two thirds smaller than the mature flesh-worms met with in the muscles of the cat. These had migrated from the intestines, and after working their way between the fibres of the muscles, had become encapsuled—the cap-

sules being perfectly transparent. Herbst and Virchow have found the flesh-worms both in the mesenteric glands and in the mesentery, and therefore, presumably, *in transitu* between the intestines and the muscles. All the phenomena described occurred within the space of a single month; and even as early as three weeks after feeding, Virchow found the young brood equal in size to those administered at the commencement. The genesis, development, and migrations of these flesh-worms are thus proven to be astonishingly rapid. Dr. Thudichum has also very recently verified these experiments; and at the conversazione of the British Medical Association, held at Downing College, Cambridge, on 4th August, 1864, he exhibited the parasite, *living*, in various stages of development, which he obtained from the muscle of a rabbit infested with them, and also from some pork chops (*Brit. Med. Journal*, August 13, 1864).

Thus the *T. spiralis* has been shown to be a bisexual parasite, producing its young alive in the intestines of the animal whose muscles it may infest, either in the free or in the encapsuled state. So long as it remains in the capsule it is immature and non-sexual, and so far they are harmless; but should they become free, they grow larger, and may become mature, and so give rise to others, if the death of the animal they inhabit does not occur.

In the mucus of the intestines the mature *Trichinæ* find a suitable place for growth and breeding, their progeny finding their way to the muscles, where they eventually become encysted; and their favorite haunt there seems to be the small muscles of the larynx (ZENKER).

According to Virchow's conclusive testimony, all these phenomena occur within the space of a single month; and, in his experiments, even as early as three weeks after ingestion, the young were found to equal in size those that he administered at the commencement. The genesis and migrations of *Trichinæ* are therefore astonishingly rapid, and probably without parallel in this class of parasites (COBBOLD).

Since the discovery by Leuckart of the round worm, of which the *T. spiralis* is the immature condition, since the case recorded by Zenker, and since the more complete knowledge that has been acquired by experiments, of the wonderful migrations of the young *Trichinæ*, attention has been especially directed to the possibility of the *trichinatus disease* in man being more common than was anticipated.

In December, 1860, Professor Wunderlich met with a case of prolonged fever, which did not correspond in its course with any of the well-known specific fevers. The patient was a butcher. He eventually got quite well, and so far negatived the diagnosis of *acute tuberculosis* which had been made. A second butcher, from the same establishment, came into the hospital with the same symptoms of high fever, with immense depression; but the course of the disease again did not correspond with any of the known fevers. Here, as in the first case, the muscles were particularly implicated, but in a less degree. There was not only muscular pains, but absolute soreness of the muscles on pressure. This man, too,

eventually got well. A third and a fourth butcher, from the same house; were also taken ill with similar severe febrile symptoms, but they were not seen by Wunderlich.

These men had been killing a number of pigs; and, as is the custom, they ate of the raw flesh. Eight men so ate, and four of them were afterwards attacked with these anomalous but severe febrile symptoms. Unfortunately, none of the pork had been preserved, and the possibility of *Trichinæ* existing in it had therefore not been proven. Moreover, none of these men died, and no evidence of the parasite existing in their muscles was obtainable. But looking to the undoubted fact that the use of the raw meat brought on the disease, and to the great probability that the wanderings in large numbers of the *Trichinæ* will produce these symptoms, Professor Wunderlich deems himself justified in thinking that there are some grounds for considering these febrile attacks to have been due to *trichinatus disease*. That individuals enjoy good health although the muscles are infested with the encapsuled *Trichinæ* is now well known from the number of cases that have been seen in dissecting-rooms. Cases are also referred to by Mr. Curling, of its being recognized in the muscles of men killed by accident, when engaged in severe manual labor (*London Med. Gazette*, Jan., 1838; also Turner in *Edin. Med. and Surg. Journal*, 1860, p. 209). The distinguished teacher of clinical surgery at Berlin, Professor Langenbeck, related to the Medical Society there, in 1863, the case of a man from whom he had recently removed an epithelial cancer situated in the neck. During the operation the *platysma myoides* exhibited a singular appearance, which, on careful inspection, was found to arise from the presence in the muscle of innumerable dead *Trichinæ* contained in calcified capsules. On inquiry, the following facts were elicited: In the year 1845 there was a "church visitation" (whatever that may mean), in which eight persons took part, and of these, seven afterwards sat down to a breakfast consisting of ham, sausages, cheese, roast veal, and white wine. In the course of three or four days every one of the seven persons was seized with diarrhoea, pains in the neck, œdema of the face and extremities. Of the seven, four died, and the three who survived (among whom was the man operated upon eighteen years afterwards by Professor Langenbeck) remained ill for long afterwards. The suspicion arose that poisoning, through the agency of white wine, had taken place; and an investigation was made, but without any result. The innkeeper, however, at whose house the breakfast was given, being still under suspicion, was obliged to give up his business and emigrate. The importance of such a case in all its forensic aspects cannot be over-rated; and it becomes an important subject of inquiry whether some of our cases of death from suspected but unproved poisoning may not be due to *trichina disease*, which is now known to be much more prevalent than has hitherto been supposed, both in this country and in Germany.

Very recently attention has been again awakened on the subject by an occurrence almost tragical.

About the middle of October, 1863, there was a festive celebration at Heltstädt, a small country town in Prussia, near the Hartz Mountains, numbering from 5000 to 6000 inhabitants. One hundred and three persons sat down to an apparently excellent dinner, mostly men in the prime of life. Within a month more than 20 persons had died, and more than 80 persons were then suffering from the fearful malady, while those who were apparently unscathed were in hourly fear of an outbreak of the encapsuled flesh-worms.*

The dinner had been ordered at a hotel, and it was arranged that the introduction to the third course should consist of "Röste-wurst." The sausage-meat was therefore ordered at the butcher's the necessary number of days beforehand, in order to allow of its being properly smoked. The butcher, on his part, went to a neighboring proprietor of pigs, and bought one of two pigs from the steward of the pig-farm. The steward unfortunately sold a pig which his master intended should not be sold, because it was not considered to be in good condition. Nevertheless, for this time at least, the butcher got "the wrong sow by the ear." The ill-conditioned pig was the one that was killed and worked up into sausages. These were duly smoked and delivered at the hotel; and after being toasted before the fire (so as to be warmed through merely) they were served to the guests at the dinner-table.

On the day after, several persons who had eaten the dinner were attacked with great irritation of the bowels, loss of appetite, great prostration, and fever. The number of persons attacked rapidly increased; so much so, that great alarm was felt in so small a town lest an epidemic of typhoid fever was about to set in.

But one of the physicians at last conjectured that some poison must be at the bottom of the outbreak, and an active inquiry into all the circumstances of the dinner was instituted; and when the muscles of the calves of the legs of some of the sufferers began to be affected, the descriptions of Zenker's case (already described) was at once remembered. The remnants of sausages, and of pork employed in the manufacture of them, were examined with the microscope, and found to be literally swarming with encapsuled flesh-worms. From the muscles of several of the suffering victims small pieces were excised, and under the microscope they were seen to be charged with *Trichinæ* in all stages of development. It could, therefore, no longer be doubted that as many of the 103 persons as had dined together and partaken of the "Röste-wurst" were affected with trichinous disease by eating the trichinous pork, the flesh-worms of which had not been killed by the smoking and toasting. On the contrary, the subdued heat of toasting would rather foster their vitality.

This catastrophe awakened sympathy and fear throughout the whole of Germany. Most of the leading physicians were consulted in the interest of the sufferers; and some visited the neighborhood

* [See the report of this outbreak of Trichiniasis, by Dr. Thudichum, in the Seventh Report of the Medical Officer of the Privy Council, London, 1864.]

where most of the affected patients were. None could bring relief or cure. Case after case died a slow and lingering death, by exhaustion from nervous irritation, fever, loss of muscular power, inflammation of the lungs, or of organs essential to life. The cases have been observed with great care and chronicled with skill. All the features of the remarkable disease have been registered in such a manner that hereafter there can be no difficulty in recognizing the disorder.

The disease begins a few days after eating the meat in which there were *Trichinæ*, with loss of appetite, and, almost without exception, with diarrhœa and fever; œdema of the eyelids; also pain, or at least painful sensation of weakness, in the limbs; œdema of the joints; difficulty in moving the tongue; profuse clammy perspiration; and those patients who do not become convalescent die either unconscious, with symptoms of typhoid fever, or, in a few cases, remain conscious to the end, complaining of inability to breathe freely.

[There would appear to be three stages in the disorder: (1.) Until the progeny are born; lasts from four to eight days; the symptoms, general discomfort, gastric trouble, especially after eating, vomiting, and diarrhœa. (2.) From the beginning of their wanderings, till they are encysted in their permanent resting-places; the symptoms, fever, and profuse perspirations; œdema of the face, sometimes extending to the whole body, inflamed conjunctivæ, photophobia, and pain on moving the eyes, especially on looking upward; scanty, high-colored urine, diarrhœa and tenderness of the abdomen; rigidity, swelling, weariness, formication, and severe pain on moving the muscles, first, of the neck and back, next of the arms and thighs, then of the fore-arms and legs; breathlessness and hiccough come on when the respiratory muscles are infested, hoarseness and loss of voice on invasion of the laryngeal muscles, and difficulty of mastication and deglutition on the muscles of these functions being affected. The sufferer lies on his back, with his legs drawn up, unable to move or speak. The fever continues or increases; typhoid symptoms set in; there are much exhaustion, meteorism, restlessness, and sometimes delirium; great wakefulness appears, often very early, and fainting fits. Lobular pneumonia may intercur, with pleural effusion. This stage may end in death within four to six days, though commonly it lasts three to five weeks. (3.) The symptoms lessen on the reappearance of the urinary secretion, but stiffness of the muscles may last for some time, and baldness and epidermic desquamation may take place. In milder cases, there is a feeling of general discomfort, restlessness, lassitude, vertigo, loss of appetite, thirst, wakefulness, or disturbed sleep, lumbar pain, swollen face, and diminished urine. Its attacks are said sometimes to be very insidious, and the patient who has not been severely ill, dies suddenly from pneumonia or peritonitis. Children are said to suffer less than adults.]

The only important symptom of typhoid fever absent in the disease is the enlargement of the spleen; and it is very probable that some of the so-called epidemics of typhoid fever in former days were caused by the propagation of *Trichinæ* in the human body.

[There should be no difficulty in the differential diagnosis of the trichin-

ous disease and typhoid fever. There is nothing in common in the distinctive symptoms of the two disorders. The epistaxis, the characteristic physiognomy, the pain and gurgling on pressure in the right iliac region, the rose-colored eruption of typhoid fever, are all wanting in the trichinous disease; while in the latter, the early troubles of the digestive functions, followed by œdema of the face, and severe muscular pain, especially on motion, with the breathlessness which often worsens to threatened asphyxia, present a series of symptoms in regular and close sequence, corresponding to the successive epochs of the larval production in the intestines, and the migration to and abode in, the muscles, which happens in no other disorder.]

Since the disease has been known (about five years ago), a great many cases have been observed in Germany.

[In 1860, Dr. Zenker recognized the disease in Dresden, and on examining the ham and sausages that had been eaten by the persons affected, found them infested with trichinæ. In 1859, 1860, and particularly in 1862, many cases were noticed at Blankenbourg, chiefly amongst the soldiers. In 1862, of 60 attacked, 2 died (SCHOLZ). Two cases were seen by Wunderlich in Leipsic in 1861; and Wagner describes 5 cases which occurred there in 1863 (*Archiv der Heilkunde*, 1864). Landois met with 12 cases in the island of Rügen in 1861, and Wentzel with 20. In that year, at Cosbach, 3 persons of the same family, who had eaten of fresh pork, and in whose muscles Zenker found trichinæ, were affected. In the same year, 300 fell ill with the disorder in Magdebourg, and 2 died. In the summer of 1862, at Calbe, 30 persons, in a population of 1200, were attacked—9 males, 25 women, and 4 children, and 8 died—1 male, 6 women, and 1 child. In the spring of 1862 there was an outbreak at Plauen, in Saxony, and several died (BÖHLER). In the autumn of 1863 the Hettstädt epidemic occurred, already described. There was an outbreak at Hedersleben in 1865 (300 cases and 40 deaths); at Zittau, in 1866 (57 cases); and at Görlitz (80 cases and 1 death).

But few cases of the trichinous disease have been recognized in the United States. The first cases reported are believed to be those of Dr. Joseph Schnetter, of New York; 2 cases after eating underdone pork-steaks; neither were fatal (January, 1864); and 5 cases and 1 death (February, 1864), of persons who had eaten raw ham, in which trichinæ were subsequently found.* About the same time, Dr. Voss, of New York, had 4 cases on board one of the Bremen steamers, then in the harbor. Dr. Voss verified his diagnosis by cutting down on the deltoid muscle of one of the affected persons, and removing a portion for microscopical examination; it proved to be filled with trichinæ.† Dr. J. R. Lothrop, of Buffalo, has reported a case.‡ Nine cases have happened in 1866 in one family at Marion, Iowa, and been reported by Dr. Joseph H. Wilson.§ About the 5th of May, six persons in the family of Mr. Bemiss, of that place, were taken ill, with the characteristic symptoms of the trichinous disease, which was not, however, at first recognized, and the disorder

* [*Observations on Trichina Spiralis*. By John C. Dalton, M.D. The Transactions of the New York Academy of Medicine, vol. iii, 1864.]

† [Dalton, *l. c.*]

‡ [*A Treatise on the Principles and Practice of Medicine*. By Austin Flint, M.D., 1866.]

§ [St. Louis Medical Reporter, July 15, 1866; Chicago Medical Journal, August, 1866.]

was looked upon and treated as typhoid fever. On the 14th of May, three other members of the family became similarly affected. It appeared that towards the end of April a couple of smoked hams had been bought, and from that time until the 5th of May all the nine had eaten of it sliced raw, and all had been taken ill in from five to ten days. Five of the nine died. Two post-mortem examinations were made, and trichinae were found in large numbers, and very active, in the muscles, in the lungs, and spleen. All the organs appeared healthy to the eye. It is stated that one of the family ate some of the meat "rarely done, and was affected but slightly," and another, when well cooked, who was unaffected. No portion of the offending ham was got for examination, but it was shown that some of it had been given to a healthy sow, who died on the first of June, "with all the symptoms of hog-cholera, and on some of her meat being examined, it was found swarming with trichinae." Dr. H. Ristine, of Marion, Iowa, has also reported six cases,* happening in the same country, in four families living in the same neighborhood, all children, their ages ranging from seven to seventeen years. It appears that on the 25th of April, 1866, they had all eaten chips of raw ham. "On the 27th, they were, most of them, seized with diarrhoea, followed in two or three days by the other characteristic symptoms. In the oldest girl, the order of succession was reversed, the muscular pains preceding the diarrhoea." The "counterpart of the eaten ham, put up in brine, was examined and found to contain trichinous cysts." The same meat, it is stated, was eaten by the family of the owner, when well cooked, and with no bad effects. We have no data at present to estimate the degree of prevalence of trichinous disease amongst the hogs of this country.

The symptoms of the disorder in the pig are said to be loss of appetite, a hoarse voice, and aversion to movement, particularly to running, and when this is attempted there is dragging of the extremities (Cobbold). Still it is positively asserted that the animal may be infested, and yet show no signs. Cobbold mentions an instance where a pig appeared remarkably healthy, and yet the butcher who ate his flesh died of the disease. Delpech says, in his report to the French government, "It is rare that any symptoms are spontaneously developed in the infected animal which would lead to any suspicion of the disorder: it has the appearance of perfect health. The butchered meat too looks well."† Dr. H. Jardine states (*loc. cit.*) that in his vicinity, the opinion prevails that the trichina spiralis exists in the flesh of animals affected with hog-cholera, the symptoms of this disorder being diarrhoea, swelling of the neck, stiffness of the limbs, debility, and cough; but the opinion has not been yet verified by microscopic examination.

The Chicago Academy of Sciences appointed in the spring of 1866 a committee of physicians "to examine into the facts concerning the supposed existence of trichinae in pork raised in this country." The results of their well-conducted observations were, that having procured and examined portions of muscles taken from 1394 hogs in the different packing-houses and butcher-stalls of Chicago, they found trichinae in the muscles of 28 hogs; from which they conclude, that in the hogs brought to that city, 1 in 50 is affected with trichiniasis in a greater or less degree; which would indicate that trichiniasis in pork is even more common in this country, or in that of the Northwestern States, than in Germany. In the

* [*The Medical Record*, New York, August 6, 1866.]

† [See the report officially made to the French Minister of Commerce by MM. Delpech and Raynal, *Bulletin de l'Académie de Médecine*, May, 1866.]

town of Brunswick, North Germany, where a most careful inspection of 19,747 hogs was made in the years 1864-65, only two were found to have trichinæ in their muscles; "the proportion being 1.10000 to 1.50 in the Chicago pork."* One of the tables of the Chicago committee shows the great variation in the number of helminthes infesting the several muscles examined. An approximation only to the number existing in a cubic inch of a given muscle could be obtained. The method adopted, was to count the trichinæ existing in several different portions of a muscle, each a cubic one-tenth of an inch in size, and to multiply the average number to a cubic inch. Of twenty-eight specimens examined with this view, only three of them contained over 10,000 to the cubic inch,—18,000, 16,000, and 15,000, respectively. The remaining twenty-five were infested to a much less degree—from 48 to 6000 in the cubic inch. It was calculated that a person eating an ordinary meal of the pork from which the specimen containing 18,000 to the cubic inch was taken, would soon become infested with not less than 1,000,000 of young trichinæ.†

With regard to the muscles of the hog which are the most common site of trichinæ, the observations of the Chicago committee do not agree with those of European observers. In Germany, the inspectors of pork are instructed to examine microscopically nine different sets of muscles, namely,—those of the diaphragm, tenderloin, shoulder, front and back of neck, extensors of the fore-arm, flexors of the leg, and the muscles of the larynx. In the trichinous-infested muscles examined by the Chicago committee, more than one-half were spinal muscles, which are not named in the German list.

In conducting an examination of the trichinatus pork, the tendinous extremities of muscles should be selected, as here they are usually most numerous. The cysts are visible to the naked eye as whitish, round, or ovoid specks, sprinkling the surface of the muscle. If a very small piece of muscle is cut off with scissors, and then torn in shreds with a needle, freeing the cysts from the flesh, and these are touched with a drop of hydrochloric acid, the lime is dissolved and the white coloring disappears; or a piece of the suspected flesh may be put into a watch glass with liquor potassæ (1 part to 8 of water), when it becomes changed to a mucus-like, clear mass, and the capsules will be seen as sharply defined minute white specks (LEUCKART). But it is always better, if possible, to use the microscope, and trichinæ not yet encysted can only be recognized by the microscope. A thin layer of the suspected flesh should be cut out with a sharp knife, and spread over a glass plate, moistened with a drop of water, covered with a thin piece of glass, and examined by a magnifying power of 50. Their intimate structure cannot be recognized with a less power than 200 (ALTHAUS).‡]

The vitality of the *Trichinæ* is not destroyed unless the meat or other substances in which they are located be subjected to the tem-

* [Chicago Medical Examiner, April, 1866.]

† [As many as 2,000,000 trichinæ have been estimated in the muscles of a man who died of the disorder, and Prof. Dalton counted in a piece of muscle (in one of Dr. Schnetter's cases) $\frac{1}{16}$ th of an inch square, and $\frac{1}{32}$ th of an inch thick, where they were in average abundance, twelve trichinæ, which would give in round numbers, over 85,000 to the cubic inch; and in the portion of muscle taken from the living subject, in Dr. Voss's case, they numbered a little over 7000 to the cubic inch (Dalton, l. c.). In one of Dr. Wilson's cases, which proved fatal, 104 trichinæ were counted in a piece of the rectus femoris muscle measuring $\frac{1}{16}$ th of an inch square, and $\frac{1}{16}$ th of an inch thick, which would give nearly 180,000 to the cubic inch.—EDITOR.]

‡ [On Poisoning by Diseased Pork: being an Essay on Trichinosis or Flesh-worm Disease; its Prevention and Cure, by Julius Althaus, M.D. London, 1866.]

perature of boiling water for a sufficient time to insure that every particle has been acted upon by that degree of heat. Salting, smoking and toasting trichinous meat, as is usually done, does not appear to be sufficient to destroy the worms in all parts of the meat.

[Thorough cooking will destroy trichinæ, and the meat must be subjected to a temperature of at least 160° Fahrenheit. If the interior of a piece of boiled or roasted pork has much of the blood-color of uncooked meat, the temperature has not been higher than 131° Fahrenheit, and there is still danger, the encapsuled trichinæ requiring a higher heat to destroy them (FIEDLER). Hot smoking for twenty-four hours will kill them, but cold smoking, though continued for several days, will not (LEUCKART, DELPECH, KUCHENMEISTER). Long and thorough salting, is said to be destructive to them; also simple desiccation of the meat, if sufficiently prolonged.

The comparative immunity from the disease in the United States is most likely gained by the habit of well cooking the meat liable to be infected, whilst in Germany, it is, by the poorer classes at least, often eaten raw. For the same reason the apparent absence of the disorder in France may be accounted for, the French always cooking their meats more than the Germans.]

Picric acid (*acidum picro nitricum*) was tried with the hope that it might be administered with success to the patient, but it failed. In trichinous pork of a pig killed with picric acid the worms were found alive (W. MULLER, of Homberg).

When this flesh-worm was seen more than thirty years ago, it was little thought that the bit of muscle sent to Owen contained the germs of a disease which might be carried in a living pig from Valparaiso to Hamburg, and then kill almost the entire crew of a merchant vessel. It has been recently related that a pig so diseased was shipped at Valparaiso, and killed a few days before their arrival at Hamburg. Most of the sailors ate of the pork in one form or another. Several were affected with the flesh-worm, and died. One only escaped being ill. Numerous cases of fever, and epidemics of inscrutable peculiarity, are now claimed by medical writers, with much show of reason, as outbreaks of the flesh-worm disease.

Professor Eckhardt, of Giessen, has obtained permission to produce the disease in a criminal condemned to die, and to try various remedies on him. (For a very interesting account of *Trichinous* or *Flesh-worm* disease, the reader is referred to the recent work of Dr. Althaus on this subject.)

For the diagnosis of *Trichinæ* in the muscles of man, Kuchenmeister has proposed to *harpoon* the muscles; but this seems a very severe operation.* Welcker believes that the best place to look for them is under the tongue, close to the frænum: in cats they can be easily seen in this situation. Whether it is so in man is not yet known (Virchow's *Archiv.*, 1861, p. 453, quoted by Dr. Parkes, *l.c.*)

* [To obtain a piece of a muscle of a trichinous patient, by means of Middeldorff's, or Duchenne's harpoon, is neither difficult, dangerous, nor painful; and the diagnosis of the disorder is made sure, at a certain stage, by direct and positive means.—EDITOR.]

[It would appear that emetics and active cathartics, if employed in the first stage of the disorder, while the parasites are yet chiefly in the alimentary canal, are of use. It is recommended that large doses of olive oil be subsequently given. The sleeplessness and copious perspirations are best relieved, according to Ruprecht, by wet packing. But some substance, which is capable of destroying the trichinæ, without damaging the bearer, is desirable. Dr. Mosler, from his experience with the disorder at Quedlingburg in 1863, is of opinion that the only rational treatment for the trichinous disease in man is benzine. His formula is: *Benzine*, f3ij; *liquorice-juice*, mucilage of gum arabic, āā f3j; *peppermint water*, f3iv. A tablespoonful to be given every hour or two hours. Dr. Mosler states that in this form benzine is well borne, and that none of his patients felt any bad effects from its use. It was largely used in the Hedersleben outbreak, but with no good results. Fiedler has shown that the pretended cures with carbazotic (*nitro-picric*) acid, reported by Friedreich, were mere coincidences, and he proposes Dippel's animal oil. Dr. Tavernier suggests carbolic acid. Küchenmeister speaks favorably, in recent cases, of equal parts of turpentine and sulphuric ether. At best they can only kill the trichinæ in the intestinal canal; when these begin to migrate, all remedial measures must be unavailing; and if recovery takes place, it is because the parasite is finally nested in its calciform cyst.]

6. The *Sclerostoma duodenale* is known to be tolerably common throughout Northern Italy; and, according to Pruner, Bilharz, and Griesinger, it is so remarkably abundant in Egypt that about one-fourth of the people are constantly suffering from a severe anæmic chlorosis, occasioned solely by the presence of this parasite in the small intestines.

"Its length is about one-third to half an inch, its width about one-twentieth of its length. Its head has a round apex, and its extremity, which is bevelled at the expense of its posterior surface, is provided with hooklets that occupy converging papillæ. The mouth contracts, to open into a thick muscular pharynx, which, widening as it passes downward, ends, after occupying one-seventh of the body, in the intestine. The sexual differences of the male and female are very interesting. Its pathological significance is chiefly due to the hemorrhage caused by these parasites, which are often present in thousands between the *valvulæ conniventes* of the duodenum, jejunum, and ileum, and not infrequently in the submucous areolar tissue. In short, the physician practising in Egypt must never forget that the chlorosis of this climate is often the result of repeated and small hemorrhages from the intestine caused by these parasites. Turpentine, as Griesinger points out, promises to be the best remedy both as a styptic and as a vermifuge" (*Brit. and For. Med.-Chir. Review*, l. c.).

7. The *Strongylus bronchialis* was first discovered by Treutler, in 1791, infesting the enlarged bronchial glands of an emaciated man. The parasite is cylindrical, slightly narrowed anteriorly, filiform, but somewhat compressed at the sides, semi-transparent posteriorly, and of a blackish-brown color. It measures from half an inch to three-quarters of an inch in length.

8. The *Eustrongylus gigas* is fortunately rare in man, though common in a great variety of animals, such as weasels. It inhabits

the kidney, destroying the substance of the organ, the walls of which become the seat of calcareous deposits.

9. The *Speroptera hominis* is furnished with a spiral tail, having peculiar marginal appendages.

10. The *Filaria medinensis*, Guinea worm, or *Dracunculus*, lives amongst the connective tissue of man and of some animals. In this situation it is only known as a female, containing an enormous quantity of young *Filaria*, and resembles a long piece of uniformly thick white whip-cord. In this country few are familiar with its appearance, or with the lesions it produces; and we therefore look for our knowledge regarding the main points in the natural history of this parasite to be furnished to us by the observer in Africa or Asia; and of whom we hope that they will fill up the gaps which still exist.

The Guinea worm is essentially a tropical parasite. It is endemic in the hot intertropical regions of Asia and Africa, extending from Egypt, about 23° or 24° north latitude, to Sumatra and adjacent islands, as far as 10° or 12° south. But it is only in some districts within these tropical limits that the parasite abounds. For example, it is endemic in Arabia Petrea, the borders of the Persian Gulf, and of the Caspian Sea, the banks of the Ganges, Upper Egypt, Abyssinia, and Guinea. Its occurrence in Guinea (although it has its common name from this place) is extremely capricious. In some districts every native who comes off to the ships seems to be affected by it; in other places in Guinea it is very rarely seen.

The *F. medinensis* is unknown in America, unless the person in whom it exists has been in the places where the *Dracunculus* is endemic. The only exception is the island of Curaçuo. It is sometimes so extensively disseminated that it has been said to prevail after the manner of an epidemic.

Although this parasite rarely causes death, still it is often the cause of great distress and loss of strength to regiments quartered in those places where it is endemic.

In the *Statistical Sanitary and Medical Reports of the Army Medical Department* for 1860, the admissions for *Dracunculus* into the hospital may be shown as follows:

I.—EUROPEANS.

STATIONS.	Average Strength.	Total Admissions.	Ratio per 1000.
Home Stations,*	97,708	28	3
Mauritius,	1,886	17	9.0
Bengal,	42,371	51	1.2
Madras,	10,696	19	1.7
Bombay,	11,388	114	10.0

* It is of course to be inferred that these men had served abroad in countries where *Dracunculus* is endemic.

II.—BLACK TROOPS AND ASIATICS.

STATIONS.	Average Strength.	Total Admissions.	Ratio per 1000.
Sierra Leone,	879	1	2.6
Gold Coast,	313	77	246.0
South China,	2611	78	26.0

It would be interesting to know how long each man was off duty or under treatment for the disease induced by this parasite. In India the average number of days which those affected with the worm remain in hospital increases progressively with advancing years. During the *first period* (18 and under 20 years of age) the average number of days under treatment—during which period each person was rendered ineffective—was 14.8; during the *second period* (20 and under 25 years) it was 16.188 days; during the *third period* (25 and under 30 years) it was 18.001 days; during the *fourth period* (30 and under 35 years) it was 22.718 days; during the *fifth period* (35 and under 40 years) it was 24.290 days; during the *sixth period* (40 and under 45 years) it was 31.620 days (EWART).

Dr. Leith, in the *Bombay Mortuary Reports*, records 133 deaths from *Dracunculus* in eight years (from 1848 to 1857). A fatal result generally takes place from hectic (LORIMER) and exhaustion, consequent on the copious discharges which sometimes follow the presence of the parasite, or from abscesses forming and bursting into the abdominal cavity (EWART). Death has followed from tetanus (Drs. Minas and McKenzie, *Trans. of Hyderabad Med. and Phil. Society*). Great destruction of tissues sometimes results from sloughing; and deep-seated inflammation may attend its existence, with the formation of abscesses and deep-seated sinuses. The death of one person is recorded by Dr. Minas at Sirsa, in whom the whole body and skin was a network of Guinea worms. As a rule, however, the patient is unconscious of the presence of the *Dracunculus* till it is matured and ready to make its exit.

The Number of Worms observed in any one individual is very various. In the majority of cases only one is present, or known to be making its exit at one time. But there are remarkable exceptions to this rule. Mr. Forbes mentions that most of those affected have had two worms extracted; but many have had four, five, and six; and when he wrote he was then treating a man in hospital in whom no less than fifteen were exposed to view, and many of these were extracted. Dr. A. Farre mentions that as many as *fifty* worms have been met with in one person. Such cases, however, are confessedly rare even in India, where *fifteen* worms is about the greatest number observed.

Seat or Locality of the Parasite.—The lower extremities are by far the most frequently affected—or rather, the parasite most frequently tends to make its exit there;—98.95 per cent. of the parasites do so.

Two cases are recorded by Lorimer which were remarkable in this respect, that one gave vent to seven and the other to thirteen parasites. In the case where *seven* parasites were extracted, there were *two* from the left foot, *three* from the left leg, *one* from the right leg, and *one* from the left forearm. In the case where thirteen parasites were extracted, *four* were taken from the left foot, *two* from the right foot, *two* from the left leg, *one* from the right leg, *one* from the right thigh, *three* from the right forearm.

Dr. Scott writes that he has known the *Dracunculus* make its appearance in the socket of the eye, in the mouth, in the cheeks, and below the tongue. Dubois records its exit from the nose, the ears, and the eyelids. Dr. Kennedy records cases in which the parasite made itself apparent in the back and muscles of the loins. One preparation exists in the Museum of the Army Medical Department, in which a great number were removed from beneath the scalp. Instances are recorded in which the worm has been found in the internal viscera. All such cases are regarded as extremely rare. It is of importance to notice, however, that both Dr. Scott and Dr. Van Someran agree in stating that the men who carry water in India, in leathern bags on their backs, are infested by the *Dracunculus* on all that part of the skin that has often been wetted; while Drs. Chisholm and Scott state that the legs of persons who walk among grass (especially during the rainy season, and particularly gardeners and agriculturists, and those who are obliged to wet themselves frequently) are at all seasons liable to *Dracunculi*. Some animals are said to be affected by the parasite. Forbes says that horses and dogs are so affected, and relates that a "tatoo" (a small Indian horse) was exhibited at Dharwar, having a *Dracunculus* protruding from its right hind fetlock. The parasite was of the usual size, and made its appearance as a boil; and no difference could be perceived in any respect in it from the *Dracunculus* which infests man. Clot Bey remarks that dogs are also sufferers; but on this head information is greatly to be desired.

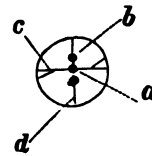
Migratory Powers exhibited by the Guinea Worm before Extraction.—Dr. Smyttan relates the cases of two officers, in one of whom the *Dracunculus* could be felt, and traced with the fingers like a cord under the skin at the top of the shoulder. By and by it made its way to the elbow, where it was equally distinct; and in a few weeks it gradually worked its way to the wrist, whence it was extracted. In the other case the Guinea worm was observed under the skin inside the biceps, and about the middle of the upper arm. It then passed round the elbow joint and down to the middle of the forearm, then back to the region of the inner condyle of the humerus, whence it was extracted. It was three months engaged in this peregrination. Dr. Paton records similar cases (*Edin. Med. and Surg. Journal*, 1806, vol. ii, p. 151); and Dr. Morehead says of his men that they had felt the Guinea worm in the thigh in the first instance, and subsequently they had been ejected from the foot. He has distinctly noticed the corded feeling of the worm below the skin, and observed that it was entirely gone the next day he examined the part. Dr. L. W. Stewart, of the Madras

Medical Service, relates a very distressing instance of this kind which happened to an officer who had already extracted a Guinea worm fifteen inches long from his scrotum. Ten days afterwards he experienced an unpleasant sensation in the posterior aspect of the left thigh. Day by day the sensation shifted lower down, till it reached the popliteal space. A few days later the sensation was experienced in the calf. Hitherto nothing was visible; but at the end of sixteen days from the first sensation in the thigh the convolutions of a Guinea worm could be distinctly traced at the outer side of the ankle-joint. Dr. Stewart now wished to cut down and extract the parasite, but the evening was too dark, and he delayed till the following morning. By the morning visit, however, the parasite had again fled, and had taken up a position in the deeper muscles of the foot. Not a trace of the worm could be recognized in the place which he had evacuated. Many abscesses now formed, and severe inflammation of the foot resulted, which confined the patient for three months before he was free of this wandering parasite. Dr. Ewart says he has seen the worm change its position from the upper part of the lateral aspect of the thorax to the groin in the course of twenty-four hours; but he has never seen the creature travel from below upwards (*Indian Annals*, vol. vi, p. 490, July, 1859).

Structure of the Dracunculus.—It is often a matter of extreme difficulty to extract the worm without breaking it, and, on account of its remarkable elasticity (for it may be extended to twice its apparently natural length), good measurements of any large number of worms are not easily obtained (Busk).

Of forty Indian specimens Ewart gives the average length at 25.25 inches, the shortest being 12 $\frac{1}{4}$ inches, the longest 40 inches. Clot Bey records their length at from 6 inches to 4 feet, in Egypt. Carter gives their dimensions in India at about 28 inches long, and $\frac{1}{4}$ th of an inch in diameter. He has dissected five. Busk gives the dimensions at from 4 to 6 feet, and $\frac{1}{2}$ th of an inch in diameter; and he has made out that it grows in the human areolar tissue at the rate of about an inch a week. H. C. Bastian, Esq., of University College, has recently read an account of the anatomy of this parasite at the Linnæan Society; and from the records of these excellent observers we have now a very complete account of the anatomy of the Guinea worm.

FIG. 22.*



The anterior end of the worm (Fig. 22) may be recognized by a "punctum" in its centre, $\frac{1}{800}$ th of an inch in diameter, surrounded by rugæ in circles, the external of which was $\frac{1}{4}$ th of an inch in diameter. Above and below are two papillæ opposite each other, with a transparent area in the centre of each. These are rather

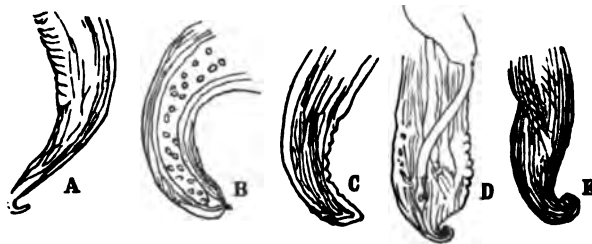
* Diagram of the head or anterior end of the Guinea worm; showing (a.) Punctiform mouth $\frac{1}{800}$ th of an inch in diameter; (b.) Upper large papillæ; (c.) One of small lateral papillæ; (d.) One of four crucial white lines meeting at the mouth, and occupying intermuscular spaces (H. C. BASTIAN, Esq.)

oval, $\frac{1}{15}$ th of an inch in diameter, with a transparent area of $\frac{1}{150}$ th of an inch. Besides these, two lateral tubercles exist, much smaller, more indistinct, and further from the punctum than the upper and lower papillæ. They are $\frac{1}{150}$ th of an inch in diameter.

It is difficult to obtain a good view of the head; for, as it is the first part to protrude through the skin, it is usually rubbed off or destroyed by the treatment adopted for extraction.

Great varieties in form are presented by the tail or posterior end of the worm (Fig. 23). The remains of the attenuated extremity of the young *Filaria*, being more or less persistent in the form of a hook or spikelet, was believed at one time to be the penis of a male; and such specimens as showed such spikelets have been mistaken for male Guinea worms. All these forms, as Busk showed, have been found in specimens containing living young ones (proligerous). All are females that have yet been found, and no males are known to exist in the human body. The strength of the tissue of the *Dracunculus* is such that a loop of the parasite

FIG. 23.*



will suspend a weight of 11½ ounces (SCOTT), and it is elastic to a remarkable degree. On opening the body, two longitudinal muscular bands are seen on the dorsal and two on the ventral aspect, running from end to end; while circular or transverse rugæ mark the whole extent of the worm; and these are approximated or apart as the worm is contracted or extended. The body of the worm (Fig. 24) contains an alimentary canal, which commences at the "punctum," and terminates in the concavity of the tail end. It is of a yellow color, nearly uniform in size throughout its extent, and in its course through the body winds several times round the genital tube (BASTIAN). No outlet has yet been detected. It is distinct from the tube containing the young (FORBES).

The genital organs consist of a large uterine sac or tube, occupying nearly the whole length of the worm, and terminating abruptly at either extremity in a much smaller tube (probably ovarian), about three-quarters of an inch in length. No vagina or vulva can be discovered (BASTIAN).

The whole extent of this uterine sac or capsule is crowded with

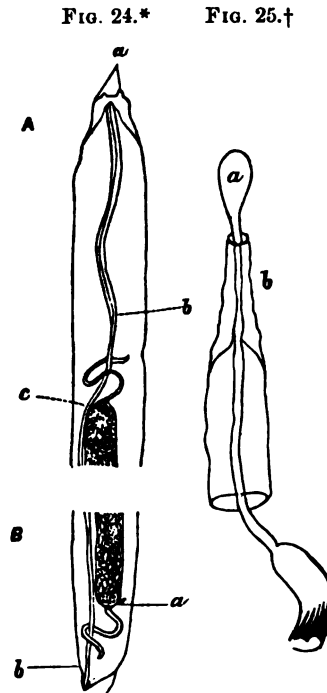
* Various forms of the caudal end of the Guinea worm.—(A, B, C) after BUSK—all of them proligerous; (D) after CARTER; (E) after GREENHOW.

innumerable young, and, with the exception of a transparent half inch or so of the worm, the whole extent of the parent seems to be a *uterus*, a *matrix*, or a *proligerous capsule*, carrying a countless offspring, to which no parturient female of any animal can be compared for productiveness; and from the fact that no inlet has ever been discovered to the genital organs, and from various circumstances, Mr. Bastian has endeavored to show that this innumerable progeny has been produced by a process of *parthenogenesis* similar to that with which we are so familiar in the *Aphis*.

If a living worm recently extracted be well lit up by an argand lamp, the hair-like filaments may be seen in motion with a good simple lens; and if a section be made across the parasite after it has been hardened in glue, the young may be demonstrated *in situ* (Fig. 26).

When the animal is mature, and presenting its head through the skin, it protrudes the extremity of the proligerous capsule through one of the small papillæ or puncta, carrying forward a prolongation of something in the form of a loose corrugated sheath (Fig. 25). It gradually assumes the form of a dilated vesicle filled with limpid fluid—the contents of the proligerous capsule—containing flocculent granular matter and young Guinea worms. Carter tells us that, if kept moist, the full-grown parent will live many hours; and in this state the young will live till the parent begins to decompose; and when the head end of the worm during its extraction may have been dried up for several days outside the wound, the remaining part with the young still remains alive. Mr. Busk says that the young survive after having undergone a considerable degree of drying up.

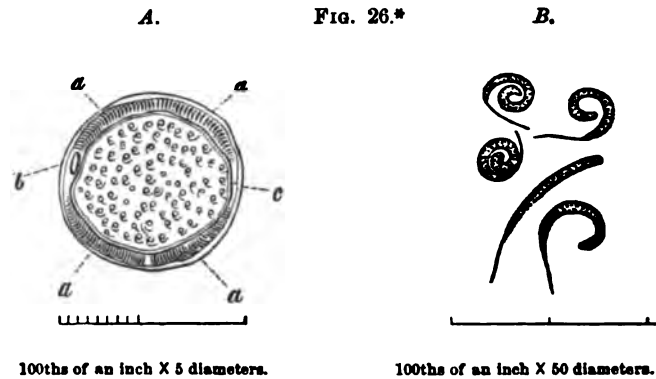
Description of the Young.—They are exceedingly numerous, and constitute the bulk of the contents of the parent's body; but are less numerous towards the tail end. Each young one may be said to consist of a body and a tail, hair-like and finely pointed. The



* A.—Anterior extremity of worm, slit open and magnified, showing, (a.) Upper and lower cephalic papillæ in profile; (b.) Junction of oesophagus with intestine, and constriction of peritoneal sheath; (c.) Anterior termination of uterus, with short ovarian tube. B.—Posterior extremity of worm, slit open and magnified in same way, showing its hook-like termination; and, (a.) Posterior termination of uterus with ovarian tube; (b.) Termination of intestine (BASTIAN).

† Anterior extremity. The ovisac (a.) is protruded, dilated, and contains young; (b.) A funnel-shaped sheath surrounding the protruding ovisac (GREENHOW).

body constitutes $\frac{3}{4}$ ths, and the tail $\frac{1}{4}$ ths of the whole length. The anterior extremity has a blunt end, with a rounded oval orifice



communicating with a cavity occupying about one-half of the whole length of the body, and terminating cœcally.

Symptoms and Life of the Guinea Worm.—As a parasite in the human body it may be studied during two periods of existence; but from the beginning to the end of its cycle of development its history embraces at least *three*, if not *four*, phases of existence or forms of life: (1.) During the first period of its existence in the human body the Guinea worm parasite is latent, residing in the connective tissue, at variable depths from the surface. During this period it does not exert any irritating influence on the surrounding tissue, as has been shown by dissections (BUSK). (2.) The second period of existence comprehends that of ripening or maturation of the worm and its progeny, when the worm makes itself felt, and begins its exit through the skin. This period is marked by characteristic symptoms. Drs. Scott, Forbes, Morehead, Lorimer, and Van Someran all agree in stating that the earlier symptoms are a pricking itching heat, which is felt at the part where the worm exists, seldom amounting to pain till after the lapse of three or four weeks. A small vesicle forms over the part, which immediately precedes the appearance of the anterior end of the worm. Dr. Scott was himself a sufferer, and writes feelingly on this point. (See *Med.-Chir. Review*, 1823.) This itching may happen before any vesicle forms; and when the vesicle forms it rapidly enlarges—so rapidly that in a few hours it attains the size of a good large filbert (LORIMER). If this vesicle is opened early, it is seen to contain a clear and limpid fluid (the fibrinous serum of irritation?); but if untouched for a day or two, its contents become turbid, and sometimes bloody, from the rupture of the proligerous sac, and the dis-

* A.—Appearance of transverse section of adult Guinea worm, as seen throughout the greater part of its length.—(a, a, a, a.) Sections of the *four* longitudinal muscles; (b.) The intestine flattened, and lying along the edge of one of the longitudinal muscles; (c.) Walls of the uterine sac, often adherent to the parietes of the body. B.—Young of the Guinea worm more or less spirally curved (BASTIAN).

charge of the young *Filaria* amongst the serum. These greatly add to the irritation, so much so, that when the cuticle is removed, an angry-looking ulcer is exposed, in the centre of which the parasite may be seen presenting itself, *with a thin transparent tendril about an inch in length hanging from its point.*

After the appearance of the vesicle or blister, it is sometimes weeks before the worm protrudes itself.

The contents of the blister, when turbid, are a discharge from the tube of the animal; as Wilkins, of 4th Light Dragoons, first surmised, and as shown afterwards by the independent observations of Forbes, who found that the best way to procure the young Guinea worm for microscopic examination was to lay open this vesicle before the delicate membrane of the proligerous capsule burst. After the escape of the serum from the vesicle, the delicate transparent membranous tube or *cul de sac* is sometimes protruded from the extremity of the worm; and if cold water is *gently* poured in a *constant stream* upon this protrusion, the dilatation and protrusion increase, till an innumerable quantity of young is ejected from the ruptured orifice of the dilated tube. Forbes says that he has often repeated this experiment; and in one instance the transparent tube was again retracted within the limb, after three emissions of young Guinea worms. On the following day the tube was found again protruding as before; and the same result (namely emission of young) followed the gentle application of the stream of water. The animal will emit its young daily in this way for some time; and when it ceases to emit them, it is then time to begin the extraction of the parasite (FORBES).

The Period of the Year when Dracunculus is most Prevalent.—This seems to vary considerably in different parts of India, and the probable causes of these differences are of great interest in regard to the origin and spread of this parasitic affection.

At Madras and its vicinity Guinea worm annually appears with greater prevalence during the hot season (LORIMER), comprehending February, March, April, May, and June. At Dharwar and its vicinity the admissions to hospital for Guinea worm generally commence in April and May. At this time water is scarce, every tank is dried up, wells yield a scanty supply, and the natives are obliged to remain at the bottom of the wells by turns, till the required supply is obtained; and when the monsoon sets in (rainy season), the admissions gradually increase through June, July, August, and September. The increase of the disease amongst soldiers or residents seems to advance with length of residence, generally during the rainy season. In the Bombay and Matoongha districts the admissions to hospital begin in May or June (irrigation of fields by the natives being common at this time), but it chiefly prevails during the rainy months of June, July, August, and September, and is rare after October (SMYTTAN). Dubois, a missionary at Sattimungalum, says that its annual endemic prevalence in the Carnatic villages is in December, January, and February, during which time more than half the inhabitants are affected. Dr. Morehead's experience at Kirkee and vicinity gave March, April, May, June, and July, as the months of gradual increase and prevalence; and September,

October, November, December, and January, as those of comparative exemption.

In the Bheel districts Guinea worm begins to increase in frequency in February; it is *four* times as frequent in March, and *six* times as common in April, as in February. It reaches the monthly maximum of prevalence in May. It prevails to a great extent in June, and continues to be common throughout the monsoon months of July and August. During September, October, November, December, and January, it is least of all prevalent. The half of the year comprising the hot and rainy season is, therefore, the period when *Dracunculus* abounds, abruptly commencing with the initiation of the former, and terminating more abruptly still with the exhaustion of the monsoon in September (EWART).

All the records agree in assigning to this parasite—(1.) An annual periodic recurrence; (2.) Periods (annual) of progressive increase and subsidence; (3.) A probably fixed latent period of residence in the connective tissue—a period of incubation—of not less than twelve months (LORIMER, MITCHELL); or of twelve to eighteen months (BUSK).

The Guinea worm never makes itself manifest in the human body before the second season of residence in the places where it is endemic, a complete season being requisite to mature the worm.

There are some remarkable cases which fix the period of incubation of the Guinea worm in a very decided way. For example, in some excellent remarks on this subject by J. Mitchell, Esq., in the supplement to the *Madras Times*, of December 18, 1861, and January 13, 1862, it is related of a gentleman, well known to be extensively acquainted with natural history, that when he was travelling in the Northern Circars, the tents were pitched near a tank of bad repute. He was accompanied by five friends, who, against his advice, bathed in the tank. Each of these five persons subsequently became affected with the Guinea worm. In the Indian annals many accurate accounts are given which fix the period of incubation at about twelve months.

Geological Features of Locality and Soil where the *Dracunculus* is Endemic.—Evidence of a circumstantial kind tends to connect the parasite with something geologically characteristic in the *soil, mud, moisture, or water* of the places where the parasite is endemic; yet information is still very imperfect on these points. Morehead believes that all the districts where *Dracunculus* prevails are composed of the secondary trap rock—*i. e.*, of igneous formation, as in the villages of the Deccan and Northern Concan, where the parasite is indigenous. In the country between the Western Ghauts and the sea-coast, where the parasite is rare, the soil is a conglomerate iron-shot clay, of a red color.

Chisholm's investigations on this point led him to the conclusion that the districts where Guinea worms abound (*i. e.*, in man) are of volcanic origin, with an argillaceous soil, holding much moisture, impregnated with salts or percolated by sea-water. Dr. Carter's evidence as to soil is, that the parasite abounds where the soil is a decomposing trap, of a clayey consistence, and of a yellow color.

Every regiment which has occupied the lines of the 24th Regiment at Secunderabad, "near the large tank called the 'Hausen Saughur,'" has suffered from the *Dracunculus* (LORIMER). The cause of the disease exists *in or near* the lines at that place; and the soil is marshy which borders on the tank. The experience of the 19th, the 4th, 5th, 1st, and 35th Regiments of Native Infantry, all fix the locality of the Guinea worm germs to be "*in or near these lines.*" For example, the 19th Regiment arrived at Vepery on the 20th May, 1838. It had been free from *Dracunculus* for five years before: twelve months after its arrival twenty-eight cases of Guinea worm appeared, and several cases amongst the followers and children. The 45th Regiment occupied the same lines previous to the arrival of the 19th Regiment; and the disease appeared amongst them at the same season of the year, and after twelve months' residence. The Guinea worm had not been amongst them for many years before. At Perampore (in the 1st Regiment, N. I.) it manifested itself, after twelve months' residence, in March, April, and May. For many years previously Guinea worm had been unknown in the Regiment. Those who suffer most in cantonments are those who use water of the filthiest kinds.

On the authority of Scott, Smyttan, Chisholm, and Duncan, Guinea worms are said to have been found in the earth or soil, and that they have been dug out of moist earth. There can be little doubt, however, but that the worms so found were specimens of the *Gordiaceæ*.

In some form or another, therefore, the Guinea worm has an existence in moist earth and mud; and it is probable that the hair-like worms found by gardeners in India coiled up together may be the young *flaria* of the Guinea worm in sexual congress; whose progeny, as *Zoöspirms*, or as filiform female worms (like the Tank worm of Carter), make their way into the body. It is known that the *Gordius aquaticus*, when young, enters the bodies of large water beetles, and at a certain stage of life it leaves its abode in the beetle and goes into the water, where it becomes a variety of *Tank worm*. It appears that there are white and brown *Tank worms*—nay, that there are no fewer than seventeen species of minute *Filaria* (CARTER, MITCHELL); and some say that all *Tank worms* are white at first, but become black after a time in the water (GUNTHER). Observations are greatly wanted on these points. According to observations collected by Pallas and quoted by Vogel, it appears that even in Europe thread worms like the *G. aquaticus*, common in stagnant water and moist earth, can in certain cases infest the human subject (*De Infestis Viventibus intra Viventia*, p. 11).

The most obscure and incomprehensible parts of the history of this parasite are—(1.) The phase of its existence and that of its young after it leaves the body of man; and (2.) The future life of the young, and their sexual differentiation.

The parasite may be removed in several ways by surgical interference—either by cutting down upon it; or, after it begins to show itself, to commence winding it on a stick, gently pulling a portion of it out every day. But there is a natural termination to all dis-

eases; and it is a fair subject of inquiry as to what becomes of the *Dracunculus* if left to itself, and its expulsion unaided by art. How would it be expelled, and what becomes of the progeny? Is it probable that they would ever be placed in circumstances where they could lead an independent existence, becoming sexual, and multiplying their kind?

In reply to these questions it is to be observed that there are undoubted examples of the *spontaneous evolution or expulsion* of the Guinea worm. Scott once observed about five inches of the worm to start suddenly out, firm, elastic, and spirally twisted like a cork-screw, showing evidence of resistance to a progressive force from behind. So firm was the parasite that it supported itself for a little time perpendicularly to the limb. It is only when the animal dies that great mischief happens to the part where the parasite is. Then and there it acts as a foreign body; but alive it does not cause disturbance (John Hunter *On the Blood*, 4to, 1794, p. 208). The part first protruded is the head; and its future progress, though slow and invisible, becomes in time very obvious (Scott).

As an example of its spontaneous evolution or expulsion, Dr. Forbes relates that on one occasion eight Sepoys were admitted with Guinea worm, and all of them had a characteristic vesicle on the ankle. These vesicles were opened on the fourth or fifth day. The loose skin was cut away with scissors, and a stream of cold water was poured daily on the part. Under these circumstances the young were daily ejected from the proligerous tube of the parent parasite, and continued to be so for fifteen to twenty days. After this time a watery fluid only was emitted, without any young, but sometimes containing particles of a white flaky appearance, which continued two or three days longer. The Guinea worm then became flaccid, and was discharged spontaneously, without pain or swelling. The only exception was in one case, where the worm was constricted by the pressure of a band of areolar tissue, which led to retention of the young, and sloughing.

Dr. Kennedy relates an anecdote which has an interesting bearing upon the spontaneous evolution and the probable future of the Guinea worm after expulsion. "In 1791, when marching up the Ghauts with a Sepoy battalion, an African stepped out of the ranks and requested permission to go to a rapid running stream of water near by, in order to relieve himself, after his own fashion, of a worm in his ankle. The man unbound a bandage from his foot, loosened the worm (of which a part was extracted) from the cloth round which it was secured, and plunged his naked foot into the current of the stream. The constant but gentle force of the running water was sufficient to stimulate the worm to come forth, and it was extracted almost immediately." Another custom has been recorded by Dr. Lorimer, which illustrates the spontaneous evolution, and points, at the same time, to the probable future of the Guinea worm. He says, "Many people belonging to the bazaars in the vicinity of the lines, affected with the parasite, came for the express purpose of extracting the worm to the same tank where the men of the regiment bathe. The people so infested swim about

in the water with the worm hanging loose, drawing the limb quickly backwards and forwards through the water, and from side to side, till expulsion is effected." The natives do not believe that they get the parasite from bathing in the water.

In these and similar cases the parent, being carried away in the stream, finds a place to die, and so gives freedom to her immense brood of young. The water seems congenial to the parent Guinea worm, and sooner than anything else induces her to leave her position in the human body, and so to extricate herself, perhaps by stimulation of the muscular structures. This water method of extraction was also recommended by Dr. Helenus Scott, of Madras (*Edin. Med. and Surg. Journal*, vol. xviii).

Vitality of the Parasite in Water.—It has been stated that young *Dracunculi* die in four, five, or six days if placed in pure water from well or tank (and that is the case with many animals), simply for want of food. Water *not pure* is, no doubt, the proper element for them (MITCHELL). Those artificially kept in impalpable red clay, partially covered with water, and exposed to the sun, were found alive after fifteen, eighteen, and twenty-one days, burrowing into the fine, soft and ochry mud.

Forbes experimented on two pups five or six months old. He poured down their throats water containing the young Guinea worm *Filaria*. After three minutes the first pup became uneasy, sick, and vomited; the watery part of which was found to contain the animal still alive. Four hours after this the pup was killed, when abundance of *Filaria* were seen in the mucus of the stomach and duodenum; but none showed signs of life. The other pup was killed twenty-four hours afterwards, but none were alive, although abundant in the mucus. Lorimer tried upon himself and others if the parasite could be propagated by inoculation of the young *Filaria* emitted from the parent's orifice. Five besides himself were inoculated. He naively remarks that *he is sorry to say* they did not hatch in any, although in his own case he put them in their favorite place—namely, the *foot and ankle*.

Such experiments were not likely to succeed, from the delicate nature of the young *Filaria*, and because they were introduced under unnatural circumstances. Inflammation and pus are inimical to the life of the worm. Besides, it is most probable that they enter the body in some other form. They seem to go through another stage of existence, and become sexual; for it is *only females*, and these *impregnated ones*, which are found in the body of man. The progeny of the sexual *Filaria*—and the impregnated females only of that progeny—would therefore seem to be the *Dracunculus* of man.

Dr. Ewart, in his able paper on the vital statistics of the Meywar Bheel corps, writes as follows: "I am inclined to believe that Guinea worm is propagated by a female and impregnated *ZoöspERM*, and not directly from either the young of the full-grown female Guinea worm or from tank worms" (*Indian Annals*, vol. vi, July, 1859).

Examination of Water, Mud, and Tanks.—In the months of August and September, 1837, Dr. Forbes examined several of the tanks in

the vicinity of Dharwar, and found the mud on their banks, and in half-dried beds, abundantly supplied with animalcules (*Filaria*), some of them very much resembling those produced by the Guinea worm when infesting the human limb. Their vermicular motion in the water is exactly the same; their general appearance is the same; and they are active and equally numerous. The point of a penknife inserted into the mud will raise up abundance for examination. They are most numerous where the water assumes a variegated appearance, with a pellicle floating on its ochry surface; and the fine, soft, impalpable mud just above water-mark contains most, and the best time to find them is about three or four o'clock in the afternoon. Two kinds may generally be detected in the soft mud: one kind is seven or eight times the size of the Guinea worm young *Filaria*, the other exactly resembles them.

The larger one may be the more mature form of the progeny after becoming sexual.

The smaller one may be the first generation born of that sexual progeny—whose females, being fecundated, enter the body of man in this young and minute condition.

Dr. Carter had medical charge of a school containing nearly 400 children. "One morning a case of Guinea worm in a child little more than four years old was reported to him. There having been only two cases of this disease in the school during the previous eight years, Dr. Carter, who had before noticed the resemblance of the aquatic *Filaria* of Bombay to the larva of the Guinea worm, was led to make inquiries, when he learned that the child was the son of the sergeant of the Industrial School situated about three miles off, and had been only a little more than three months in the school. Upon further inquiry he found that the sergeant's wife had then a Guinea worm in her ankle, and that twenty-one out of fifty boys had been affected with Guinea worm during the past year. Some boys had had as many as five extracted, and ten more were then suffering from the disease, all of whom had been in the school more than a year. None of those who had been less than twelve months in the school had been admitted to hospital on account of Guinea worm.

"The boys were living in an embanked inclosure that had been taken in from the shore, the fourth side of which was formed by a cliff of the mainland, on which resided the sergeant and his family. In this inclosure were two small tanks, ten feet square, sunk in decomposing trap, one being six feet, the other three feet deep; the first furnished drinking-water, in the latter the boys bathed. The sergeant also obtained his bathing-water from these tanks, but the drinking-water from a well at some distance.

"The tanks contained *Conserve*; and Dr. Carter states that every small piece as large as a pea contained twenty or thirty of the tank worms. He examined the *Conserve* of the tanks at the Central Schools, where there had been no cases, or only two in eight years, and failed to find the worm after the closest scrutiny. Hence he argues, and with apparently good reason, *No Tank worm—No Guinea*

worm; but that persons who bathe in water in which the former is found may expect to have the latter.

"Dr. Carter further states that the Industrial School is situated near an old artillery barrack, now in ruins and overgrown with weeds, which had to be abandoned in consequence of the havoc made among all ranks, officers as well as men, by this fearful parasite" (MITCHELL, *l. c.*).

The habit of the tank worm is to bury itself under any organic debris that may be in the water in which it is found; and if it be disturbed, it will immediately seek a hiding-place, nor rest until again covered. This implies that its proper habitat is the bottom of tanks, wells, or other reservoirs, among the decayed and decaying organic matter. It may be assumed that the water-carriers referred to by Dr. Morehead were Army Bheesties, who as such probably had access to good puckah wells (Dr. Morehead having found that Guinea worm was not more common among them than among other people), and as the tank worm, habitually resident in the mud at the bottom, would only be disturbed when the water became very low, and would get back again to its retreat if possible, the fact of water-carriers being as little affected with Guinea worm in the upper part of the body as other people does not carry so much weight as at first it would seem to do, and as it would in reality if the tank worm was in the habit of swimming at the surface like many other aquatic animals. It has not been said that the worm finds its way into the body by any of the natural cavities of the body, such as the alimentary canal. On the contrary, it is supposed that the water may be drunk with impunity, as known by experience, and from the experiments of Forbes already noticed.

The young *Filaria* can work its way into a proper receptacle by its pointed extremity, "which is a long cone, ending in a point so inconceivably fine that the point of a cambric needle is a large marline-spike in comparison with it." But notwithstanding its exceeding tenuity, it appears tolerably rigid, and as the proper receptacle referred to is one of the sudoriparous ducts, a ready-made aperture exists for a distance quite long enough to contain so small a creature; and it is by no means inconceivable to one who has seen its active exertions, that it should be able thus to hide itself in a foot or leg kept for some time in the water. It is unnecessary, perhaps, to do more than allude to the well-known native custom of going into a tank to take water. In these tanks water-carriers may often be seen standing for five or ten minutes at a stretch, chatting and washing themselves. They of course stir up the bottom mud, and, if the tank worm be there, and is the origin of the Guinea worm, they certainly afford it every opportunity to effect a lodgment—the instinct of the parasite directing the effort. One circumstance which makes this the probable mode of entry is that natives are much more subject to attack than Europeans.

Thus the evidence is very strong which refers the entrance of the parasite to bathing or lying on moist places where the tank worms abound.

Greenhow states that the sepoy of the Maiwara battalion bathe and drink the water of a well sunk in the limestone rock, which generally contains about twenty-eight feet of water, clear and sweet; while the prisoners of the jail at Beaur use similar water from another well; but they never bathe, which the sepoys do every day. The result is that *Dracunculus* is much more prevalent among the sepoys, compared with the prisoners, in the proportion of three to two. Again, amongst "Puchallies" the numbers affected are four times as great as among the men of the regiments. The former frequent the tanks more than the men of the regiments.

Generation and Propagation of the Guinea Worm.—The following periods may be recognized in its natural history: (1.) It is probably got by bathing in tanks or places where the young and impregnated females abound. (2.) A period of maturation in the human body takes place. (3.) A time favorable for extraction comes, when the animal seems to seek delivery from its imprisonment, to fulfil a new law of its existence. The adult animals perish annually. It is necessary they should die, that the young may live; and, indeed, the Guinea worm of the human body is not adapted to live. It has no functional arrangements for life.

Men being exposed to the cause about the same time, the period for extraction will arrive about the same time in all, but with just sufficient variation (as to time) as to suggest the idea of contagion (Scott, *Med. and Surg. Journal*, vol. xvii, p. 99). But the idea of contagion or infection from one man to another (as Bruce, McGrigor, and Paton wished to establish) is quite untenable. The evidence is all the other way.

In Paton's cases on board Her Majesty's ship "Cirencester," from 30th May, 1805, to 9th August of the same year, the origin of the disease is quite traceable to the preceding July and August, when the ship lay in Bombay harbor (*Med. and Surg. Journal*, 1806, vol. ii, p. 151).

Sir J. McGrigor's cases in the 88th Regiment, and the absence of Guinea worm among the artillery on shipboard, related in his medical sketches, were not fully investigated. We have no account of the water supply previous to embarkation. Afterwards he wrote a paper, or rather an account of the sickness in the regiment from all diseases, in the *Edin. Med. Journal*, vol. i, p. 270, and from this it appears that the regiment had been quartered in the Fort of Bombay, which is partly surrounded by a wet ditch; and several months after leaving this place most of the cases of Guinea worm occurred. Bombay is well known to be extremely infested with Guinea worm.

Moseley is reported to have said that "there is as much foundation for believing *Dracunculus* to be contagious as that a thorn in the foot is contagious." As observed by Rudolphi, the parasite is known to occur in persons who have neither eaten nor drunk in the countries where it is endemic, but who have exposed themselves to its moisture and its mud. The moisture contained in native canoes is sufficient to have carried to a ship off the coast

the germs of the Guinea worm, which find their way into the seamen of the ship, who are in the habit of going into these canoes with bare feet.

Negative evidence, which would attempt to show that tank worm does not exist, cannot be received. Most of the examinations on which such negative evidence rests have been imperfect; having been made with instruments confessedly imperfect, and perhaps by men not accustomed to use the instrument. I speak only of written and published statements, and on the authority of Dr. Lorimer.

Problems for Solution.—Forty years ago Dr. Scott suggested that a patient and careful investigation of soils and waters ought to be made wherever *Dracunculus* is known to be endemic, and especially the soil round brackish wells and the beds of tanks. Morehead, in 1833, recommended that the following points be attended to, namely: (1.) Geological structure of the ground and nature of the site generally; (2.) Nature of soil, wells, and well-water; (3.) Nature of rocks through which wells are sunk; (4.) Abundance or scarcity of water; (5.) Seasons of increase or decrease of the disease; (6.) Opinions of natives.

The occurrence of Guinea worm is sometimes defined by a distance of a few miles. So it is with many algæ and minute water animals and plants as to habitat.*

2. *Filaria lentis*.—Length, $\frac{2}{10}$ ths to $\frac{6}{10}$ ths; width, $\frac{1}{80}$ th of an inch. The body is thick posteriorly, filiform, and ending in a pointed tail, transparent, and partly coiled up in a spiral form. The alimentary canal is surrounded by the folds of the oviduct.

This *Filaria* (*F. lentis*) is very imperfectly known, and the female only has been seen. It was detected by Nordmann in the liquor Morgagni of the capsule of a crystalline lens of a man whose lens had been extracted for cataract by the Baron Von Gräfe. In this instance the capsule of the lens had been extracted entire; and upon a careful examination half an hour after extraction, there were observed in the fluid two minute and delicate *Filariæ* coiled up in the form of a ring. One of them presented a rupture in the middle of its body (probably made by the extracting needle), from which rupture the intestinal canal was protruding. The other was entire, and

* My friend, H. C. Bastian, Esq., M.B., of the London University, London, has recently furnished a most interesting account of the anatomy of the Guinea worm to the Linnæan Society, and has been kind enough to furnish me with drawings of his observations; and he writes to me as follows: "Since I saw you last I have discovered several species of Carter's 'tank worms' in soft mud, &c. (at Falmouth); that is, small *Nematoids*, agreeing in almost every respect with those found by him in Bombay. The more I see of these, the more thoroughly am I convinced of the undoubted relationship existing between them and the Guinea worm, coinciding as they do in their anatomy even to minute details, and in many respects where there is a salient distinction between the anatomy of the *Dracunculus* and that of the *Ascarides*. One which I sketched to-day had an exsertile, rigid, sharp-pointed œsophagus.

"The great difficulty in the theory is to account for the fact of the localization of the disease, whilst these animals are probably so widely spread; and I suppose it is one particular species which is limited in its diffusion; but I suspect that many of those others will hereafter be discovered as parasites in animals or vegetables. The *Vibrio tritico* I have examined, and find it to be a worm essentially similar; and Dr. Cobbold tells me that he has found a long thread-like worm in the subcutaneous tissue of the back of a water-bird. The whole question wants working out."

measured about $\frac{1}{10}$ ths of an inch in length. It presented a simple mouth, without any apparent papillæ, such as are seen to characterize the large *Filaria* which infests the eye of the horse; and through the transparent integument could be seen a straight intestinal canal, surrounded by convolutions of the oviducts, and terminating at an incurved anal extremity (OWEN, p. 64).

A *Filaria oculi* vel *lachrymalis* has been described as not uncommon among the negroes on the Angola coast, where it is called *loa*; also at Guadaloupe, Cayenne, and Martinique. Its length is $\frac{1}{10}$ ths to $\frac{1}{10}$ ths of a line. It is a filiform, slender worm, pointed at one end, obtuse at the other, tolerably firm, and of a white-yellow color.

The parasite has been considered a *Strongylus* by some, by others a young Guinea worm, and by others as an *Oxyuris vermicularis*.

Treatment of those Infested by the Round Worms.—The habitat of the *Ascarides* being for the most part a collection of mucus, the means used for their expulsion is generally some purgative medicine, as two grains of *calomel* and ten grains of *jalap*, or as many of *scammony*, given two or three times a week. The purgative ought in no instance to be given oftener; for if the purging be continued, the intestine is weakened, and more mucus secreted; so that the secretion which harbors them is increased. In weakly children small doses of Epsom salts will ultimately effect the same object, and with less distress to the patient. Many persons place great confidence in *calomel* as a medicine capable of destroying them; but it does not appear to act beneficially except as a purgative, and consequently it is an auxiliary, and not by any means the most valuable part of the treatment.

The *Oxyurides* or small vermicular *Ascaris*, being situated so near the rectum, enemata have at all times been much used in the treatment of these cases; and injections of oil have been much commended, especially of castor oil, olive oil, or sweet oil. But these animals will live from thirty-six to forty-eight hours in castor oil. Indeed, very little benefit has been derived from any such local treatment. Warm water injections tranquillize the intestine, and give more temporary relief than anything else. The *Oxyurides* are killed by cold; but it may not always be safe to throw a cold injection into the colon of a child. But if the child is otherwise a vigorous child, small injections of very cold water may be cautiously administered, with a few drops of *ether* or of *alcohol*; and injections of the following bitter substances have been found very useful in the treatment of the *Ascaris vermicularis*: Three or four ounces of a strong infusion of *quassia* repeated three or four times, or a similar quantity of *lime-water*, have been found of service. At the same time it is also well to administer internally some bitter medicine;—for example, half an ounce (or any dose suitable to the age and strength of the child) of compound decoction of *aloes*, taken in the morning fasting, once or twice a week; and three ounces (or other suitable dose) of infusion of *quassia* may be taken every morning that the *aloes* is not taken.

Chloride of sodium, to the extent of an ounce in a pint of *quassia* infusion, has also been found a useful injection; so also has an enema

composed of *aloes*, *carbonate of potash*, and *mucilage of starch*. But whatever local remedies are used, it is necessary to attend to the general health, which usually is at fault. The digestion is generally slow and imperfect, the secretions from the mucous membrane of the alimentary canal being abnormal. For this condition, small doses of the extract of *nux vomica*, with *sulphate of iron*, in extract of *gentian* or *aloes*, or in rhubarb or colocynth pill mass, taken twice a day, will be found of great service. Santonine may be of service in some cases.

From what has been already written, it will be seen how important it is, in the treatment of all these diseases, to take every means of utterly destroying, by burning or by chemical agents, all debris or excreta which may be passed by patients suffering from these parasites, and also how necessary it is to look well to the purity of all water supply used either for the purposes of food, drinking, or bathing, and to the quality of pork or bacon, in connection with the *trichina spiralis*.

ACCIDENTAL PARASITES.

Of this provisionally named class, several forms of which have been named, the occurrence of "PENTASTOMA CONSTRICTUM" in the human body as a cause of painful disease and death may be given as an example.

The author had two portions of lung and three portions of liver, each containing an unusual parasite, sent to him from Jamaica, in August, 1865, for the Museum of the Army Medical Department at Netley. Staff Assistant-Surgeon Edward Barrett Kearney, Esq., is the donor of the specimens; and from his history of the patient's fatal illness, the following account has been drawn up:

On the 11th of January, 1865, private Isaac Newton was admitted into the hospital of the 5th West India Regiment, at Up Park Camp, Jamaica, for an attack of *tonsillitis*. He was an African, enlisted about eight months previously from the slave depot at Rupert's Valley, St. Helena, where all slaves captured in slave-ships are kept until disposed of. He appeared to be about twenty-one years of age, and of a thin, spare habit of body.

On admission the tonsils were inflamed and enlarged, but not ulcerated; and there were aphthous ulcers about the tongue. He suffered from headache and pain across the back.

On the morning of the 14th he complained of great pain in the abdomen, which became tympanitic. His tongue was clean, but vividly red at the edges and tip, and it felt dry to the touch. The skin was very hot and dry and harsh, and his pulse 100. The pulse continued to increase in quickness; sordes soon began to appear about the mouth and teeth, and the tongue became furred and cracked. Large moist crepitation was heard over the whole surface of both lungs. He became low, and disinclined to be spoken to, and by six o'clock in the evening his mind appeared to be confused. He passed his urine and his stools involuntarily in bed.

On the 15th there was no improvement in his condition, and at ten o'clock at night he appeared to be in much the same state as before, and the bowels were confined.

On the 16th he appeared livelier in the morning, the skin cooler, but still dry. He was thirsty, and sordes were still about the lips and teeth. The conjunctivæ of both eyes were stained of a vivid yellow color.

About nine in the evening he became suddenly worse. His pulse became very weak and almost indistinct, the skin cold, the countenance sunken, and covered with a copious perspiration. He appeared to be sinking, and he died at half-past ten that night.

Post-mortem Examination Fourteen Hours after Death.—The general appearance of the body was that of emaciation, with yellowness of the conjunctiva.

Thorax.—The subcutaneous areolar tissue over the chest and abdomen was of a deep yellow color. The *pericardium* contained about four ounces of deep amber-colored fluid.

The *Heart* was large and pale, but its substance was otherwise normal, and its valves healthy. The *Lungs* were both highly congested; and when cut into, a bloody frothy fluid exuded in quantity. The substance of both was very friable and yellowish in color. "On the anterior surface of the *right lung* and near the edge of its lower lobe, one or two yellow specks appeared. They were about the size of a spangle, and when cut into, worms were seen regularly encrusted in its substance." On the posterior surface of both lungs there were numerous adhesions of long standing.

Abdomen.—The *Liver* was very large, extending into the left hypochondrium. "Its surface was dotted over, both posteriorly and anteriorly, with about twenty or thirty yellow specks similar to those seen in the lung." The hepatic substance appeared paler and rather more soft than natural.

Stomach.—It was distended with air and fluid, containing about a pint and a half of a dark-green colored fluid. The mucous membrane was congested in patches, in the stomach, and along the whole tract of the intestines. There was no ulceration, and no appearance of *Tæniæ*, either continuous or in proglottides, could be discovered. Other organs were healthy.

Description of the Parasite and the Lesions it Produced.—Fig. 27 represents a small portion of the lung, with the little worm, seen at

FIG. 27.



a, curled up in its cyst. The pleura has been removed, so as to expose the "rings," "markings," or "constrictions," which are characteristic of the body of this parasite. The pleura was opaque, and considerably thickened, probably from the irritation of the parasite.

The appearance of the parasite on the surface of the liver was exactly similar to that in the lung, and therefore it is unnecessary to give another drawing; and wherever the serous covering

of the organ was sufficiently transparent, the constrictions of the parasite could be seen distinctly shining through.

Fig. 28 represents two specimens of the parasite removed from their cysts. They are of the natural size, and one of them, *a*, is much shorter than the other, the constrictions being closer together: *a* measures five lines in length, *b* measures about eight lines. In diameter they are about one line. About 20 to 23 rings or constrictions can be counted on the elongated body, at tolerably regular intervals, and somewhat spirally arranged.

FIG. 28.



Fig. 29 represents the two specimens of the parasite slightly magnified (about three diameters): *a* is the shorter; *b* and *c* are the posterior and anterior aspects of the longer of the two worms. The head end appears compressed, so as to be flat and square-shaped at the end. It is seen to be marked with five spots on the anterior aspect, as shown at *c*. The posterior aspect of the flattened head, as shown at *b*, is comparatively smooth. The elongated body is rounded, and the caudal end terminates in a blunt-pointed cone. The constrictions appear like folds of the outer covering of the worm, each fold overlapping the one which follows, from the head to the tail. The body of the parasite is rounded, and not flat, as the tape-worms or cysticerci.

FIG. 29.

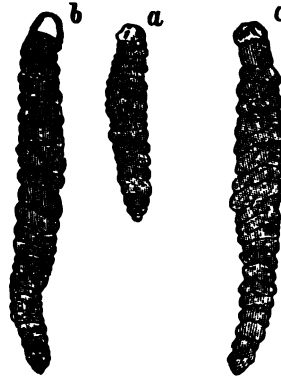


Fig. 30 represents the anterior aspect of the flattened head end (*cephalothorax*) of the parasite. It is so highly magnified as to show the nature of the five spots or marks shown in Fig. 29.

The dotted lines from *a* and *b* point to two pairs of hooks or claws—one pair on each side of a pit or mouth, *c*. The points of the claws indicated by *a* are seen nearly in profile; those at *b* are directed more towards the observer. These claws appear to be implanted in socket-like hollows or depressions, surrounded by much loose integument. These socket-like hollows appear to be elevated on the summit of the mass of tissue which lies underneath the folds of integuments surrounding the base of the hooks. These parts are regarded as the feet of the parasite, and the hooks are the foot claws.

The pit or mouth (indicated by the dotted line to *c*) is of an oval shape, the long axis of the oval lying in the direction of the length of the worm. The lip or outer margin of the pit is marked by a well-defined thin line. There are no spines nor hooks on the integument of the elongated body.

From the description and the drawings here given it will be seen that the parasite corresponds in its specific characters with the larval condition of the "*Pentastoma constrictum*." It belongs to the family *acanthotheca* of Diesing, and has no structural connection with

the true helminth parasites found in the bodies of man and other animals.

The parasite now described, when compared with the descriptions of *pentastomata* given by Frerichs, Cobbold, and other ob-

Fig. 30.



servers, demonstrates clearly that at least two species of *pentastomata* infest the human body; the *Pentastoma constrictum* being by far the larger, the more dangerous, and fortunately the more rare, of the two species. It is also still more satisfactory to know that, as a human parasite, neither of the two species has ever been detected in this country; and according to the researches of Dr. Cobbold, it is only in the encysted or larval conditions that the *pentastomata* are met with in the human body. As an embryo it becomes encysted. The cyst is composed of condensed connective tissue, and is lined by layers of loose flakes, which are evidently the remains of repeated castings of the skin of the parasite; and during the intervals of these successive moultings the worm makes considerable growth so as to reach the size in which it is finally found. In this pupa or larval condition it occurs in the solid organs of the abdominal and thoracic

cavities of man in certain geographical districts in Europe, Egypt, and the West Coast of Africa; and much more frequently in various herbivorous animals, such as the sheep, deer, antelope, peccary, porcupine, guinea-pig, hare, rat, and domestic cat (COBBOLD). In all these animals, and in man, the larvæ usually occupy cysts immediately underneath the serous covering of the liver and the lungs; and Dr. Cobbold mentions that he has occasionally found the *Pentastoma denticulatum* free in the cavities of the abdomen and pleura of animals.

Our knowledge of the natural history of these parasites is mainly derived from descriptions of the *Pentastoma denticulatum*, the larval or sexually immature condition of the *Pentastoma tanoides* of Rudolphi. We are told by Frerichs (*Clinical Treatise on Diseases of the Liver*, vol. ii, p. 276) that Pruner was the first observer who pointed out, in 1847, the existence of the *pentastoma* as a parasite in the human subject. On two occasions he found *pentastomata* in the liver of negroes at Cairo. He does not seem, however, to have deter-

mined accurately the nature of the parasite he observed; and he also subsequently found two specimens of the worm preserved in the Pathological Museum at Bologna, which had been removed from the human liver (COBBOLD). Bilharz has since repeatedly detected in the livers of negroes at Cairo the parasite discovered by Pruner in 1847. Bilharz and Von Siebold made this parasite the subject of careful study; and they recognized in it a variety of *pentastoma* quite different from that which prevailed in some parts of Germany. They gave this new variety the name of *Pentastoma constrictum*—the parasite which has proved fatal in the case whose history Dr. Kearney has sent to me from Jamaica. It is the form of *pentastoma* endemic in Egypt, and hitherto it has only been found in the African negro. It differs from the *Pentastoma denticulatum* (the larval form of the *Pentastoma tenoides*), "in not being furnished with any integumentary armature of spines, and in its being a much larger worm" (COBBOLD, p. 402). The *Pentastoma constrictum* seems to be from eight to twelve times larger than the *Pentastoma denticulatum*, and therefore is all the more dangerous from its actual size (nearly an inch long); and when it occurs in great numbers, as in the present instance, it cannot fail to prove an extremely irritant "foreign body," when it escapes into a serous cavity like the pleura or peritoneum—a mode in which it seems to cause death. The latter parasite (*P. denticulatum*) has been fully described by Frerichs, and figured by him in his *Atlas*, plate xi, fig. 9, as endemic in Germany in the human liver—in which organ it is considered to be far more common than the echinococcus.* Frerichs, however, regards the *pentastoma* endemic in Germany as devoid of clinical importance, because it does not give rise to any functional derangement. Not so, however, is such the innocent history of the *Pentastoma constrictum* as it affects the Negro; and after the history of the case now given, the clinical importance of this parasite cannot be disregarded.

As to the mode in which it tends to cause death, the evidence in this case, from symptoms and post-mortem examination, seems to point to *pneumonia* and sudden collapse from *peritonitis*. The author is able also to verify this point in the pathology of this parasitic disease still more clearly from a preparation which has been in the Museum of the Army Medical Department since 1854, but the nature of which he could not understand till the history of the case now published was so thoughtfully furnished by Dr.

* "In Germany," says Frerichs, "the *pentastoma* was first found in the human liver by Zenker, in 1854: it occurs, however, not only in this gland, but also in the kidneys, and in the submucous tissue of the small intestine (WAGNER). The parasite is by no means rare with us. Zenker, at Dresden, succeeded in finding it 9 times in 168 autopsies (or, according to Kuchenmeister, 80 times in 200 autopsies). Heschl, in Vienna, met with it 5 times out of 20 autopsies; Wagner, at Leipzig, once in 10. According to Virchow, it is more common in Berlin than in Central Germany. During six months at Breslau, I (Frerichs) met with it in 5 out of 47 dead bodies. As a rule, there is only one present; in rare cases there are only two or three. It presents the form of a somewhat prominent nodule, from 1 to 1½ lines in length, which is formed by a firm, fibrous capsule, easily detached from the surrounding parts. The animal lies coiled up in the interior of this capsule" (*On Diseases of the Liver*, vol. ii, p. 276).

Kearney, together with the specimens of the parasite *in situ* which he sent.

The preparation in the Museum, which has hitherto been a puzzle to all who have examined it, consists of four pieces of liver (Fig. 31, *a, b, c, d*), and which appears in the Catalogue with the following description:

"Portions of liver, containing numerous small cysts (evidently some jointed entozoön), taken from Private George Sutton, 1st West India Regiment, who died at Bathurst, Gambia; and for the history of the case reference is made to the annual report of sick and wounded from that station, dated March, 1854."

FIG. 31.



The author is now able to identify this preparation as an example of lesions produced by the *Pentastoma constrictum*. At *a*, in the cyst where the larva has been, there is contained the debris of integumentary exuviae; at *b* the head end of the parasite is seen peering out of an ulcerated opening in the serous covering of the liver. The edge of the opening is rounded and indurated, as if a good deal of local irritation had been maintained at the part previous to penetration of the serous covering. At *c* the ring-like constrictions of the parasite are seen shining through a very thin portion of serous membrane; and the portion of liver at *d* represents an empty cavity—whence one of these larvæ has passed out, probably into the peritoneum. The cicatricial-like contraction and puckering of tissue in the vicinity shows that considerable irritation has existed previous to the exit of the parasite.

We have no information as to how the *Pentastoma constrictum* finds its way into the human body, as an embryo and subsequent larva. Although in this instance the negro was stationed in Jamaica, it is most probable that he had the germs of these parasites within him when he left his native shores in some part of Africa; and that this parasite is neither endemic in St. Helena nor in Jamaica. Reasoning from what is known regarding the propagation and development of the *Pentastoma denticulatum*, it is probable that the ova with the contained embryos are introduced into the human stomach along with uncooked vegetable food (fruits or salads), in regions where the mature animals are endemic. From the stomach the embryos, escaping from the ova, bore their way, and find a resting-place in the liver or other solid viscus, exactly like the embryo of the tænia. In solid organs (like the lungs and liver) they become encysted, and undergo the pupal transformations so well described by Leuckart and Cobbold in the case of the *pentastoma* endemic in Germany.

The drawings which illustrate this paper were made by Staff

Assistant-Surgeon Dr. Humphrey C. Gillespie, from the preparations which are now in the Pathological Museum of the Army Medical Department at Netley.

LARVA OR GRUB THE EXCITING CAUSE OF BULAMA BOIL.

My friend Dr. Albert A. Gore, Staff Assistant-Surgeon on the West Coast of Africa, has kindly favored me with the following account of this parasite and the disease which it induces:

"This small larva or grub is of a white color, a line or two in length, and is the exciting cause of a boil occasionally seen in the Island of Bulama and its neighborhood (Fig. 32). When magnified under a low power (Fig. 33), it appears to be divided into a series of joints, and covered with minute bulbous hairs. On the anterior division are placed four or five red spots (*b*), and from either side project two hollow suction tubes (*a, a*). The posterior extremity seems to be terminated by a blunt hook. In applying a higher power, the bulbous hairs turn out to be a number of beautiful black hooklets (Fig. 34), which have a very pretty appearance on the white surface. A faint outline of a central cavity can be discerned. The hooklets are directed anteriorly.

"**Symptoms, Treatment, &c.**—Attention is first attracted to the part by feeling an extreme itchy sensation. On examination, a small red pimple is seen. After awhile a small serous discharge oozes from its centre, which sometimes seems to pulsate. If allowed to progress, it becomes a regular inflamed boil, very painful, and often causing an erysipelatous blush, with inflammation in the neighboring lymphatics, and tenderness of the glands to which they run. On the evacuation of the small abscess in the boil these symptoms gradually disappear, but a persistent red mark remains at the original seat of the disease. The treatment consists in poulticing until the little animal appears, when it can be withdrawn. If allowed to suppurate, it must be treated in the usual way of a boil under similar circumstances. The natives put in a mixture of salt and palm oil, which takes out the little grub.

"**Etiology and Pathology.**—This little worm cannot be the *chigoe* or *Pulex penetrans* of the West Indies, although it may result from the ova deposited by some similar aphanipterous insect. The *chigoe* chiefly attacks the toes or intervals between them, and causes a series of painful ulcers. It is also of a black color. This small grub is white, causes a boil or two in any portion of the body, most commonly in the thigh, arm, or abdomen; it is sporadic, although endemic. They have one symptom in common—viz., the extreme itchiness. But this is complained of in nearly every case of disease resulting from insect or other living organisms."

FIG. 32.*

X

FIG. 33.†

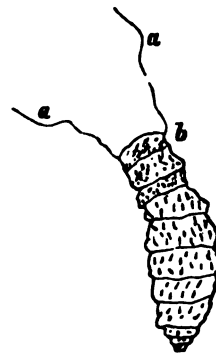


FIG. 34.‡

▲ ▲

* Natural size of larva or grub, the exciting cause of Bulama Boil.

† The same grub magnified by a low power: (*a, a*.) Hollow suction tubes; (*b*.) Fine red spots. The other dots on the body are hooklets.

‡ Two of the hooklets highly magnified. These drawings were furnished by Dr. Gore.

SECTION II.—EPIZOA—*Animals living upon the Skin and Hair; and the Pathology of the Lesions with which they are associated.*

The animals which are associated with diseases upon the skin are of three kinds:

1. The first kind belong to the family of "lice" (*Pediculidæ*), of the class *Insecta*, amongst the *annulose* or *articulated* animals. They belong to the order *Anoplura*, all of which are parasites and destitute of wings. They undergo no regular metamorphosis, as most other insects do; but in their growth to maturity they shed their skin a certain number of times, which may to some extent explain the irritation and forms of lesions to which they give rise. Almost all animals—man, quadrupeds, birds, and reptiles—are liable to be infested with these parasites; and were it not for our instinctive feelings of disgust with regard to them, as opposed to our notions of cleanliness and propriety, the study of their forms and habits is of considerable interest to the pathologist. Upwards of 500 species of lice have been described—universally diffused over the animal kingdom in different climates of the world. Their superabundance upon the persons of the human race are associated with some severe lesions of the skin; and authentic cases are related of death from lousiness.

2. The second kind of animals associated with diseases of the skin belong to the family of "mites" or "ticks" (*Acaridæ*), of the class *Arachnida* or spiders, amongst the *annulose* or *articulated* animals. Some of these are free, others are parasitic, and vary somewhat in their structure accordingly. Those which live a parasitic life have the mouth in the form of a sucker. Such are the "ticks," which fasten upon dogs, cows, sheep, horses, and other animals. They bury their suckers so deep in the skin that it is impossible to detach them without tearing the skin to which they fix themselves; and they multiply so rapidly that oxen and horses attacked by them have been known to die of mere exhaustion. The harvest ticks (*Leptidæ*), one species of which, the *Leptus autumnalis*, well known as the harvest bug, is common in autumn in grass and herbage, from which it gets on the body of man, and, though exceedingly small, produces extreme irritation of the skin. One only of these parasitic *Acari* has been found to live entirely in the skin of man—the *Sarcoptes galli* vel *Acarus scabiei*—producing the disease called the itch.

3. A third form of animal is found inhabiting the sebaceous sacs and hair follicles of the human skin—namely, the *Demodex folliculorum* (Fig. 35). By some naturalists this parasite is referred to the *Acaridæ*, but by recent investigations it is considered to be more nearly related to the *Rotifera*, its

FIG. 35.*



* *Demodex folliculorum* (after DR. ANDERSON).

parasitic habits causing it to resemble some of the numerous forms of the *Crustacea*. It is not known to cause disease.

The parasitic lesions and diseases with which these *epizoa* are associated may be described as follows:

LOUSINESS—*Phthiriasis*.

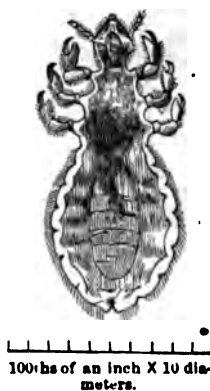
Definition.—*A morbid state in which lice develop themselves to such an extent that a pruriginous eruption is produced (Prurigo pedicularis). The skins of persons liable to constitutional skin diseases in which watery or secreting eruptions (such as eczema) prevail, are those most favorable for the development of lice.*

Pathology.—Five forms of lice infest the skin of man. One variety is met with on the hair of the head—the *Pediculus capitis*; a second variety infests the other hairy parts of the body, but especially the pubis; and hence its name—*Pediculus pubis*; the third form lives on the general trunk of the body—the *Pediculus corporis*; a fourth is the *Pediculus palpebrarum*; and a fifth the *Pediculus tabescentium*.

The first four species, although they live in close proximity to one another, yet strictly limit themselves to the regions mentioned. In a clinical point of view the *P. corporis* is the most important.

1. *The Body Louse* (Fig. 36) is of a whitish color, and varies from half a line to two lines in length; the body elongated and the abdominal portion broad, its margins lobulated and covered with little hairs. The thoracic portion is very narrow, and carries three legs on each side. The legs are hairy, jointed, and terminate in claws. The insect secretes itself amongst the folds of the clothing, and causes extreme itchiness of the skin where it comes to feed. Between the irritation of the insect, the *debris* of its exuviae, and the scratching of the skin by the patient, *papulae* arise, the summits of which being torn off, give rise to a *pruriginous eruption*, which may even become pustular. This eruption is met with most frequently on the neck, back, and shoulders, and round the waist, the parts most tightly embraced by the clothing; and where the clothes are most frequently gathered into folds, between which the lice are embedded, and where they deposit their eggs, or egg capsules, which are crystalline, shining, yellowish, opaque bodies. The lice seem to multiply fastest where eruptions such as *eczema* prevail. A case is reported by Mr. Bryant as having occurred in Guy's Hospital, in which the whole of the body was literally covered with lice. The patient had been a governess, about thirty years of age; and the irritation was so great that excoriations and scabs were produced. On admission into hospital she was put into a warm bath,

FIG. 36.*



* *Pediculus corporis*—female (after ANDERSON).

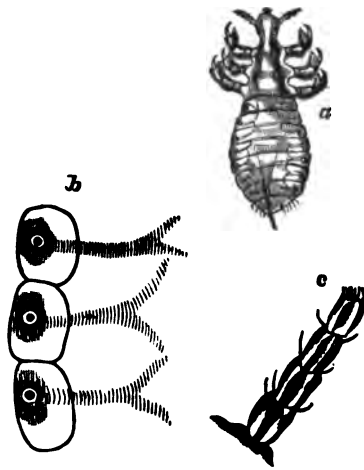
her clothes were destroyed, and every precaution taken to remove all the insects; but in two hours afterwards her body was again covered with lice, although she had been laid in a clean bed. She was again thoroughly washed, but the vermin reappeared immediately. Some of the insects and their ova no doubt remained adhering to the skin, hidden amongst the scabs of the excoriations; and they are known to multiply with a rapidity proportioned to the favorable nature of the soil afforded by the morbid condition of the skin of the affected person (ANDERSON, *l. c.*, p. 108). Bernard Valentin relates the history of a man, forty years of age, afflicted with insupportable itching over the whole body, and having his skin covered with little tuberosities. The physician, unable to assuage the itching, made an incision into one of the small tumors, and gave exit to an enormous quantity of lice of different forms and sizes. The same operation was performed on the other tumors with a similar result (quoted by ANDERSON, p. 110). The following instance is related by Dr. Whitehead, in his work *On the Transmission from Parent to Offspring of some Forms of Disease*, p. 173, and quoted by Dr. Tanner: A farmer, forty-three years of age, strong, and of sanguine complexion, contracted syphilis in April, 1840. Seven months afterwards he suffered from secondary symptoms. At the end of 1841 he became so annoyed by the presence of lice about

his person, chiefly on the trunk, that he sought again medical aid and advice. He was scrupulously clean in his habits, and had never before been troubled with these vermin. They increased in number, and produced such mental distress that fears were entertained for the integrity of his intellect. An examination of the skin showed a multitude of irritable-looking points on the front and sides of the chest, from which *nits* could be detached by lateral pressure. At this period the generation of the lice was so considerable and rapid that a flannel vest put on clean in the morning was crowded with them by the end of twenty-four hours. The usual remedies

had only a temporary effect, till *iodide of potassium* and *prussic acid*, taken internally, seemed to render the system unsuitable for the further development of the vermin.

2. *The Head Louse* (Fig. 37) is considerably smaller than the body louse just described. Its legs are larger in proportion to the size of the body than those of the *P. corporis*; and the abdomen is more

FIG. 37.*



* (a.) *Pediculus capitis* (male); (b.) Trachea and stigmata; (c.) Antenna (after ANDERSON).

distinctly divided into seven segments, separated from each other at the margins by deep notches.

They propagate with astonishing rapidity, and by their irritation produce an *eczema*, from which the fluid exudes abundantly, and crusts are formed, involving the cuticular debris or exuviae of the lice and the remains of epidermis. The hairs become glued together; partly by the fluid from the *eczema* and partly by the secretions of the insects as they deposit their ova in the capsules which they fix to the hairs (Fig. 38). These capsules are commonly called *nits*; and they adhere with great tenacity to the hairs.

FIG. 38.*

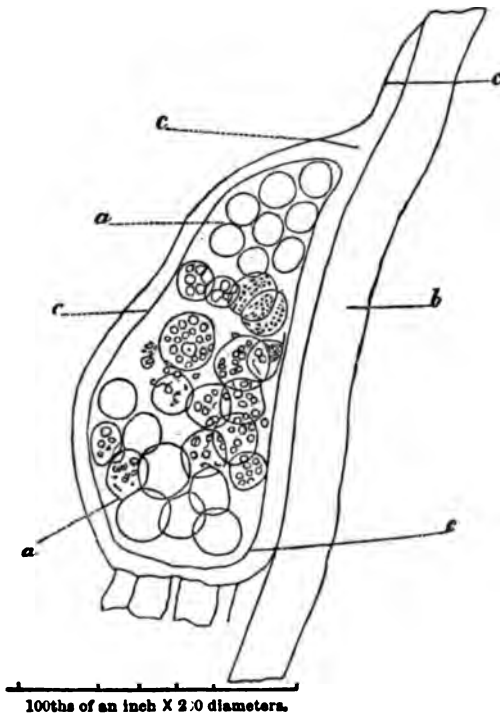
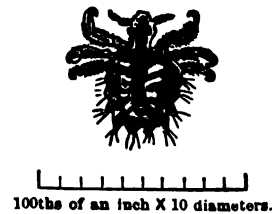


FIG. 39.†



3. *The Crab Louse* (Fig. 39) has a shield-shape, and a much broader body in proportion to its size than either of the other two forms of lice; and there does not appear to be any distinct separation between its thorax and abdomen. It has been met with on all the hairy parts of the body except the head, but more especially on the hair of the *pubis*. It does not run about like the other lice, but grasps the stems of the hairs with its fore legs, and adheres so firmly that it is difficult to remove it without pulling out the hair. The *nits* or egg capsules are attached to the hairs in the same way

* *Nit*, or Egg Capsule of the Louse, fixed to (b) a hair, by the glutinous secretion (c, c, c, c).

† *The Crab Louse* (after ANDERSON).

as on the head. Pruriginous or eczematous eruptions, which may become pustular, are the results of their existence (ANDERSON).

Treatment consists in the destruction of the insects and soothing the irritation. To accomplish the death of the parasites the following substances are efficient—namely, *sulphur, mercury, staphisagria, sabadilla, pyrethrum, the essential oils, and alcohol* (ANDERSON).

Sulphur is used in the form of vapor baths or fumigations, or the simple or compound sulphur ointment of the pharmacopœia.

Mercury may be employed as simple mercurial ointment, or by fumigations with *cinnabar*, or in solution of the bichloride, in the proportion of two to three grains to an ounce of water, to which some alcohol has been added.

Staphisagria is employed as an ointment in the proportion of an ounce of *staphisagria* with four ounces of lard, or an infusion of *staphisagria* may be made with vinegar.

Sabadilla may be used in powder or as an ointment; an ounce of lard being used to incorporate a drachm of *sabadilla*.

The hair should be cut short when lice infest the head; and a lotion of the *bichloride of mercury*, or some of the ointments above named, applied at once to cause the death of the insects. The *nits* may be dissolved away by *alcohol* or *dilute acetic acid* (HEBRA). The scalp should afterwards be repeatedly washed in warm water with soap, and the eruptions treated according to their nature.

Sometimes the nits and the debris of the lice are involved in the substance of these eruptions, so that care must be taken to kill any insects that may remain and be so hidden.

With regard to body lice, it is necessary either to destroy the clothes, or to expose them to a temperature of at least 150° Fahr., by steaming them, or ironing them over with a sufficiently hot iron, or to boil them. In extreme cases, such as those related at page 880, it has been found that a mixture of iodide of potassium and prussic acid in full doses cured the diseased state of the system which favored the development of lice in such numbers. After sixteen or eighteen doses, in the case recorded by Whitehead, the cure was permanently completed.

The *Pediculus pubis* is best got rid of by rubbing a lotion of bichloride of mercury amongst the roots of the hairs, taking care that it is brought in contact with every insect. The application should not only be applied to the hair of the *pubis*, but to that in the neighborhood, such as that of the *scrotum, perinæum, and anus*; and the application should be continued twice a day for a week at least. Mercurial ointment is equally efficacious; but care must be taken not to induce salivation: the hairs, rather than the skin, are the parts on which the lotion or ointment ought mainly to be applied.

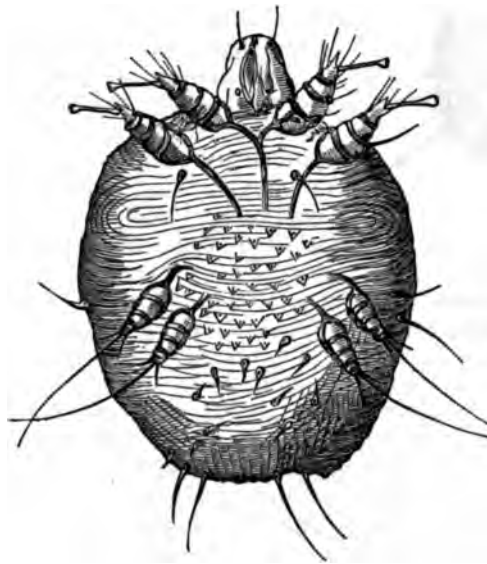
SCABIES—SYN., ITCH.

LATIN Eq., *Scabies*—Idem valet, *Psora*; FRENCH Eq., *Gale*; GERMAN Eq., *Scabies*—Syn., *Krätze*; ITALIAN Eq., *Rogna*.

Definition.—*An eruption of distinct, slightly acuminated papules or vesicles, accompanied with constant itching, due to irritation caused by the burrowing underneath the epidermis of a female Acarus (Acarus scabiei vel Sarcoptes hominis), for the purpose of depositing her eggs.*

Pathology.—The full-grown itch spider or *Acarus* is of a whitish-yellow color, and is just visible to the naked eye. The female (Fig. 40) varies in size from $\frac{1}{10}$ th to $\frac{1}{4}$ th of a line in length, and from

FIG. 40.*



$\frac{1}{10}$ th to $\frac{1}{4}$ th of a line in breadth. It is of an ovoid form, broader anteriorly than posteriorly. The anterior segment carries the head and four limbs, two on each side of the head, which are set very close to it. The head projects considerably beyond the body, is of a rounded form, and marked by a central fissure provided with mandibles. The limbs are altogether eight in number, the four posterior limbs being placed about the middle of the under surface of the body. These limbs are of a conical form, tapering towards a point. They are each composed of several jointed segments; and the four anterior limbs are each provided with a stalked sucker. The extremity of each of the hind limbs terminates in a long curved hair; and several short hairs spring beside the root of each sucker on the anterior limbs. The body is marked by numerous regularly disposed wavy lines; the dorsal surface is convex, provided with

* *Acarus scabiei*—female (after DR. T. ANDERSON).

numerous little angular spines and little round tubercles, from each of which also springs a short conical spine. From each side of the body two hairs project; and four project posteriorly; so that, including those springing from the hind legs, the posterior half of the body is provided with twelve long hairs.

The male *Acarus scabiei* (Fig. 41) is considerably smaller than the female, and the innermost pair of posterior limbs are provided with

FIG. 41.



stalked suckers as well as the anterior limbs; while the parts corresponding to the genital organs are very distinctly marked (HEBRA, ANDERSON).

It is now impossible to say who discovered the itch insect. Avenzoar hinted at the existence of an insect in the vesicles of itch; but Moufet, in his *Theatrum Insectorum*, first mentioned it in a particular manner in 1663. Hauptmann first published a figure of it, and represented it with six feet. Redi, Lorenzo, Cestoni, and Bonomo examined numbers of them, having removed them from the papules or vesicles of the skin. They discovered also the eggs of the parasite, and even observed their extrusion.

Morgagni, Linnæus, De Geer, Wichmann, and Waltz confirmed these observations; but, nevertheless, the existence of the parasite up till 1812 was still called in question. About this time, therefore, a considerable prize was offered by the Parisian Academy of Sciences for its demonstration; and M. Galès, an apothecary of the St. Louis Hospital, tempted by the reward, is said to have defrauded the Academy and gained the prize (ANDERSON). His investigations are reported to have been witnessed by many members of the Institute; but, nevertheless, he managed to conceal beneath the nail of his thumb the common cheese-mite, and having opened with a lancet the pustule of a patient affected with scabies, he dexterously produced the cheese-mite from beneath his nail, pretending to have removed it from the patient (ANDERSON). Many others attempted to find the *Acarus* in the pustules or vesicles; and the circumstance of such men as Galeoti, Chiarugi, Bielt, Lugol, and Mourouval having failed in finding the animal, occasioned fresh doubts regarding its existence. Their failures arose from having followed the cue given them by M. Galès, in searching for the parasite in the papules or vesicles. Moufet had long before stated that they were not to be found in the pustules, but by their sides. Casal made nearly a similar observation; and Dr. Adams remarks that they are not found in the vesicles, but in a reddish line going off from one of its sides, and in the reddish firm elevation at the termination of this line, a little distance from the vesicle. Seventeen years after Galès' demonstrations the Academy discovered, through Raspail, that they had been defrauded; and in 1834 M. Renucci, a medical student from Corsica, showed the physicians of Paris the mode of discover-

ing the *Acarus*, which is the same method as that which had been formerly mentioned by Dr. Adams. Since that time, as Rayer remarks, the existence of the *A. scabiei* has been placed beyond a doubt; and after the demonstrations of MM. Lemery, Gras, and Renucci (all of whom showed him the method of detecting it), Rayer has been able to extract it several times himself. Raspail has given an excellent description and figure of the parasite. M. Albin Gras enters into researches as to the share it has in producing the eruption, and he instituted experiments on the *Acarus* itself, which have an important bearing on the treatment of *scabies*. The habits and natural history of the parasite have been carefully investigated by Hebra; and Dr. Anderson has given an excellent account of these investigations, from which this description of the parasite and the disease is mainly taken.[*]

[The question whether the acarus can be transferred to man from animals seems, notwithstanding some difference of opinion amongst dermatologists, to be resolved in the affirmative. Hardy† has expressed his belief that the acari in animals produce in the human subject only temporary prurigo, requiring no treatment; but Hebra states that he has repeatedly seen scabies transmitted from animals to man in the menagerie at Schönbrunn, and he believes that the *Sarcoptes equi*, *S. canis*, *S. suis*, *S. cati*, *S. cuniculi*, of Gerlach, *S. scabiei crustosæ*, *S. vulpis*, *S. capræ*, *S. squamiferus*, and *S. minor*, of Fürstenberg, are all varieties of the same animal. He has found the acarus in the camel, the Egyptian sheep, and the ferret. Küchenmeister says that the acari of the lion and cat may be identical with the acarus in the human species; Eichstädt found in the crusts of a mangy horse acari *scabiei*; and Fürstenberg has seen them in the horse, the lion, the llama, the ape, and Neapolitan sheep.]

The discovery of the male *Acarus* is claimed by several observers. According to Hebra, it was first discovered by Danielssen and Boeck in Norwegian scabies. According to Dr. Anderson, M. Bourgogne, the maker of microscopic preparations in Paris, claims to be the discoverer. According to Devergie, the honor is due to M. Lanquetin, a pupil of St. Louis.

[The Germans give the credit of the discovery to both Krämer and Eichstädt. The former found it in 1845, but published his description and drawings in 1846.

Lanquetin states that previous to his discovery of it in 1851, he had seen one before in the cabinet of M. Bourgogne, who got it from an employé at the St. Louis Hospital (*Notice sur la Gale*, 1859.)]

It is the female only which burrows in the epidermis of the human skin. All the male *Acari* go free on the surface of the epidermis, where sexual intercourse between male and female *Acari* is said to take place. When an impregnated female is placed on the

* [On the Parasitic Affections of the Skin. By T. McCall Anderson, M.D., London, 1861.]

† [Hardy, *Leçons sur les Maladies de la Peau*, Professeurs à l'Hôpital St. Louis. 2ième partie, 2ième ed. Paris, 1868.]

surface of the skin, it seeks a suitable spot to penetrate, and raising its head at right angles to the surface, it digs, burrows, or eats its way between the scales into the deeper layers of the epidermis, where it imbeds itself, derives nourishment, and goes through the process of parturition till she dies. Having found a suitable place, an egg is laid, and each day another, the animal penetrating a little further each time, leaving its deposited eggs to occupy the space previously inhabited by itself. The direction of the canal is oblique [but horizontal through the rete mucosum], the portion first formed being of course nearest the surface. As the old epidermis is thrown off, new layers of cuticle being formed from the deeper strata, the first-laid eggs are gradually thrust upwards to the surface, where they are finally extruded, while the recently deposited ova remain in the canal close to the parent female, whose instincts lead her to make the canal in such a way that her eggs reach the surface about the time the young ones are ready to come out of the shell. The newly hatched *Acar*i (males and females) having arrived at the surface, crawl about the skin, and enter into sexual congress. The females in due time become impregnated, and, like their parent, repeat the process of burrowing and parturition just described.

[It may either proceed at once to bury itself, or it may, after awhile, leave off, and begin again at a new spot. In penetrating the superficial part of the epidermis, it seems to have some trouble; its first position is almost vertical to the skin, and it supports and braces itself by the long bristles which project from the hinder part of its body. Once through the epidermis, its steps are rapid, and it is thoroughly buried in from ten to twenty minutes. It attacks by preference those parts of the skin where the cuticle is thinnest, and a favorite site is the mouth of a hair-follicle.]

The length of time which intervenes between the laying and hatching of an egg is said to be *fourteen* days; and as the *Acarus* is found to lay one egg daily, there are rarely more than fourteen eggs in one canal at a time. •

[The number of ova deposited by a single acarus varies; Hebra has seen twenty-six in one cuniculus; and Gudden* counted fifty-one in a canal, with the parent, containing ova, at the bottom. To determine the rapidity with which they are laid, and which is said to depend on the age and degree of vitality of the acarus, Gudden put on the skin of an uninfested person an acarus, which had laid already but three ova, and which had another ready to be deposited; the subject was kept all day in a moderately warm room; at the end of five days, the cuniculus was cut out and contained eleven eggs. The acarus takes six or seven weeks for its development from the ovum to its being impregnated. In about three months after the skin becomes the "bearer" of a fertile mother-acarus, a person will generally be covered with an eruption of scabies (GUDDEN).]

* [Beitrag Zur Lehre von der Scabies, von Dr. Gudden. Würzburg, 1863.]

The canals (cuniculi) [*sillons* of the French, *milbengänge* of Hebra] which the female *Acari* burrow have a serpentine shape, and vary from half a line to three lines long. Hebra, and Dr. Reid, of Glasgow, have seen them three or four inches in length; and Hebra mentions that they sometimes completely surround the wrist, like a bracelet. These canals have generally a whitish dotted appearance, the dots corresponding to the ova in the canals; and at the extremity of each canal is a little whitish elevation, which corresponds to the site of the parturient or defunct female *Acarus*. This whitish elevation is generally about a quarter of a line distant from the papule or vesicle; and the skin should be cleaned before endeavoring to detect it.

[The two extremities of the cuniculus are the "head" and "tail," the first being the entrance-point of the acarus, and is whiter than the "tail," where the animal may be found as a sharply defined roundish point, rather deep-seated.

The appearance of the cuniculus is, however, modified (1.) by site: as just described, it is found on the hands and feet, and elbows and knees; but on the other parts of the body these appearances are modified by the development of a vesicle or pustule beneath the cuniculus. The vesicles commonly begin at the head of the passage, but frequently extend beneath it, so that it lies on their roof. The position of the parasite is always beyond the area of the vesicle, or pustule, and when a crust is formed the parent animal is never found in it, though it may contain ova, and possibly be capable of propagating the disorder. (2.) Another modification is a white dotted line on the summit of a red elongated eminence; it is found in the neck and back, in tubercular elevations (CAZENAVE), and, occasionally, on the penis, fold of arm-pit, umbilicus, nipple, and on all parts of the body exposed to long pressure from sitting or lying (HEBRA). In infants the passages may present this form on any part of the body. (3.) In old cuniculi the "head" disappears, and a red spot with a white edge continuous with the sides of the passage is left, giving to them a retort shape (HEBRA).*

Their color varies with their position, and the degree of cleanliness of the subject. On the hands and feet of the lower classes they are usually black; in those who wash often these parts, white; on the penis, buttock, elbow, and knee, they are generally white. The dotted appearance, Gudden asserts, is due to the breathing-holes in the roofs of the passages, which he and Bourguignon are positive exist there, and which Hebra as positively denies. Hardy and Bazin† believe them to be due to the small black feces of the acarus; and Hebra says, and probably truly, that they are simply caused by dirt, which has so thoroughly penetrated that it cannot be removed by washing or friction.

The most frequent sites of cuniculi are the hands and feet. In women, the line of junction of the inner side of the foot with the dorsum is a common site (HEBRA). *Acari* have been extracted from the forehead (LANQUETIN,‡ HILLIER), from within the lower eyelid, from a cuniculus

* [Virchow's *Handbuch der Speciellen Pathologie und Therapie*. Dritte Band, 1864.]

† [*Leçons Théoriques et Cliniques sur les Affections Cutanées Parasitaires*, par le Dr. Bazin, Paris, 1858.]

‡ [*Notice sur la Gule, et sur l'Animalcule qui la produit*. Par Eugène Lanquetin, Paris, 1859.]

which was on its free edge, in an infant (AUZIAS-TURENNE); and Hebra found a beautiful cuniculus, containing an *acarus* and eight ova, within the urethra, about a line from the meatus.]

After the death of the *mother Acarus*, [the duration of whose life is from three to four months (GUDDEN,)] the epidermis which covered in the canal gives way, as the cuticle grows and desquamates; there is then left at first a depression, or open ragged furrow, bounded on each side by a ragged edge of epidermis; and as these edges become dirty, the remains of the canal present a dirty ragged line. Besides the *Acarus* and its eggs, numerous little oval or rounded blackish spots are seen in these canals, which are supposed to be the excreta of the parasites; and after the female has once entered its canal it is unable to recede, owing to the spines on its body, which project backwards. It therefore dies in the canal when parturition is finished. The eggs of the *Acarus* vary much in size, according to their age and development; and just before the larva has burst its shell, the egg is almost as large as the male *Acarus*. In the earliest stages the egg is very small, and filled with a granular-like matter (Fig. 42, *a*).

FIG. 42.*



It grows in the canal; and as it increases in size its contents seem to shrink and recede from the shell, and to have a distinct enveloping membrane. The bright yellow color of the embryo contrasts strongly with the clear, almost colorless, walls of the eggshell (Fig. 42, *b*, *c*, *d*). The head and legs of the embryo soon become distinctly visible, and at last the whole form of the *Acarus* (Fig. 42, *c*, *d*). Finally, the shell bursts, and the young *Acarus* escapes, leaving its shrivelled envelope (*e*) behind. The larva or young (Fig. 43) differs from the full-grown insect in the possession of two hind limbs only, in place of four. By and by, however, it casts its

FIG. 43.†



coat, and then appears with eight legs; and sometimes even the full-grown *Acarus*, with its eight legs, may be seen inside of its old

* (*a*.) Egg in the first stage; (*b*.) In the second stage, their granular contents being yellow; (*c*.) Egg in the third stage, the form of the *Acarus* becoming apparent; (*d*.) The egg in the fourth stage, the *Acarus* having broken the shell; (*e*.) Eggshell after the escape of the *Acarus* (after Dr. T. ANDERSON).

† Larva or young *Acarus scabiei*—having only two hind legs (after Dr. T. ANDERSON).

six-legged skin, and thus renders the history of its development complete.

[Gudden states that the *acarus* moults three times. According to him, the *acarus* is characterized before the first moult, not only by its six legs, but also by having only two bristles at the posterior extremity of its body, and ten of the longer spines on its back. With its eight legs it acquires also four bristles and twelve spines, and after the second moult, fourteen spines. The sexual organs are not apparent before the third moult, and the male and female differ in the number of their spines, the female retaining fourteen, whilst the male loses two, and has only twelve. While it is changing its skin it inhabits small cavities in the skin, similar to those formed by the young *acari*.

The young *acari* on leaving the shell almost directly quit the cuniculi, through, Gudden maintains, the breathing-holes in the roof, and begin to move over the body very actively, and soon imbed themselves: the passages which they form, however, are very short,—less than a line in length. Gudden says, the young *acari* penetrate the skin more deeply than the mother, and cause more irritation, their bite being often followed by a small papule or vesicle, in which exudation shows itself on the second day. Before this time, the animal has usually moved off to another part of the skin, and this makes the young *acari* difficult to detect, and the diagnosis of the disease uncertain. Gudden thinks that a diagnosis may sometimes be made, in slight cases of scabies, and in which none of the larger cuniculi can be found, by cutting off the heads of some of the papules or vesicles, and examining them by the microscope, without varnish to make them transparent. The entrance of the passage formed by the young *acarus* can then be made out; when the light comes from below the dark edge of the opening looks dark. To obtain specimens of the young animal, oil of turpentine may be rubbed over the suspected portion of the skin, which killing them, they will be found in the papules or vesicles which first appear on the part to which the turpentine was applied.]

Symptoms.—The phenomena of the eruption of scabies are more often papular than vesicular; and the markedness of these phenomena depend partly on the length of time that the person has been affected, the number of *Acari* developed, and the degree of sensibility of the skin. It is known to infect sheep and dogs (YOUATT); and therefore hair does not preclude its existence; but it seems to prefer delicate parts of the skin—for example, the inner surfaces of the fingers, and folds of the skin between the fingers, the wrists and palms of the hands, the penis in the male and nipples in the female, as well as the hips, the feet, the umbilicus, and axillæ.

Itching, increasing at night, first attracts attention, and is a characteristic symptom. It becomes general all over the body, and the scratching aggravates the eruption. The *prurigo* of itch is generally most expressed on the forearm, lower part of the abdomen, and the upper and inner part of the thighs. Vesicular eruption is most usual on or about the fingers and nipples of females; and pustules may be met with in children whose skin is delicate, especially on the hands, feet, and hips.

[Hebra would seem to bound the part played by the *acarus* in scabies

to that of a passive carrier of the disease. He is no believer in the migratory instincts of the animal; once embedded in the skin, according to the eminent Vienna dermatologist, it is quiet, and is transferred from one part of the body to another, or even to other persons, by the finger nails of the patient, who, scratching the papules or vesicles, tears them open, and digs out the young insects and the ova. In carrying out his crotchet, he goes so far as to say that in no case is the site of the cuniculi and that of the eruption the same; for whilst the former are chiefly found on the hands, feet, penis, &c., the eruption is most abundant on the anterior parts of the body, between the mammæ, and the knees, chest, abdomen, thighs—parts of the body most accessible to scratching, to relieve the vague itching felt. Hardy says, that most generally the initial site is the penis, and that it is transferred to the fingers by scratching. The wanderings of the young animal have been satisfactorily proved by Gudden and others, and he has shown, too, that on parts of the body where scratching was impossible, papules and vesicles have made their appearance, incontestably due to the presence of young acari at points quite distant from the spot where the mother acarus nested. It is true that there is no constant relation between the intensity of the local irritation and the abundance of the eruption and the number of acari; and in the pustular scabies of children there are sometimes but few cuniculi.

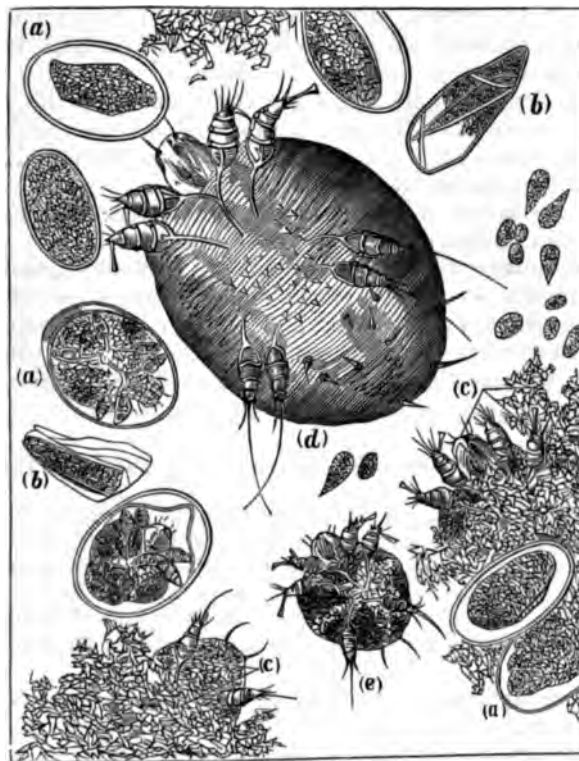
For the artificial production of the disorder Gudden proposes to extract carefully an acarus from a recent cuniculus, and then to cut off the portion of skin containing the cuniculus, and examine it under the microscope; if ova are present, it is certain that the animal will deposit others. Hebra recommends that the whole cuniculus be snipped off with a pair of scissors and applied, as a surer way.

The itch insect is sensitive to the influence of temperature, heat rendering it active, and cold making it motionless. Whenever the warmth of the skin is raised, by exposure to the sun's rays or to the fire, by the body becoming warm in bed or on exercise, its nimbleness is increased, and the consequent irritation aggravated, as those suffering from scabies so well know by the terrible itching it causes, and the relief which cooling the surface brings. As a general rule too, those parts of the body which are uncovered at night remain uninfested. Gudden mentions the case of an itch patient, a woman, who being put into a strait jacket, her hands and feet were always cold, and did not become thoroughly warm even when in bed; whilst there was a great number of acari on the trunk, none could be detected upon the hands or feet; there were a few cuniculi on the inner side of the arm, which was fixed to the body. The common site of the cuniculi, as has been observed already, is on those parts of the body most exposed to pressure. In such as sit for a long time on hard benches, as cobblers, tailors, weavers, the eruption is found on the buttocks, whilst, it is said, joiners, carpenters and bricklayers never have this part of the body affected. In women the cuniculi are found in most abundance where girdles and straps have pressed upon the skin. The skin beneath tight garters, trusses, or any band, and even a crutch, is often affected (HEBRA). The penis, and, in infants, the buttocks, are a common seat. The disease is much more frequent in winter than in summer, for in an average of eighty cases, which daily present themselves for treatment at the St. Louis Hospital, Paris, in winter, there are hardly more than ten in summer, which Hardy attributes to the poorer class sleeping and huddling more together in winter than in summer, in order to keep warm by bodily heat. Hebra, however, attaches but little importance to the influence of temperature on the habits of the acarus, or

the site of the eruption, which he refers to the irritation of the skin from pressure, or chafing.*]

There is a severe form of scabies common in Norway and some parts of continental Europe, in which the greater portion of the skin of the body becomes thickened, the natural furrows increase in depth, the pigmentary deposit is greatly augmented, and a fine white desquamation covers the surface. Here and there papules may be seen, either with dots of coagulated blood, or with whitish crusts on their summits. In these crusts portions of *Acari* and their exuviae, excrements and eggs may be found. The hairy scalp,

FIG. 44.†



covered with a crust which adheres firmly, of a bark-like consistence and yellow color, is studded with fine openings for the hairs, which are glued together. On the under surface, and in the furrows of

* [For an excellent brief of the "Recent Researches on Scabies" the reader may consult the *British and Foreign Medico-Chirurgical Review*, vol. xxxvi, 1865.—EDITOR.]

† Crust from a case of the so-called *Scabies Norvegica* which occurred in Würtzburg.—(a, a, a.) Eggs of the *Acarus* in various stages of development; (b, b, b.) Eggshells; (c, c, c.) Fragments of *Acari*; (d, d, d.) Female *Acarus*; (e, e, e.) Larva. The little oval irregular-shaped masses are presumed to be the excrement (after ANDERSON).

this crust, multitudes of *Acari* may be seen. Such are the main features of a case recorded by Bergh, of Copenhagen, and related by Dr. Anderson in his excellent little treatise already referred to. The itching was intolerable. The crusts were principally composed of the *Acarus*, its exuviae, excrement, eggs, and egg-husks (Fig. 44). A piece of the most superficial and dense part of the crust, less than half a line square, contained 2 female *Acari*, 8 six-legged young, 21 pieces of *Acari*, 6 eggs, 58 eggshells, and about 1030 pieces of excrement. "In the deepest and softest parts of the crusts, amongst the remains of deceased generations—partly in holes and passages, partly between particles of the crusts, partly on their free surfaces, masses of living *Acari* wallowed and tumbled about." The cases in which such a severe form of *scabies* has occurred have been characterized by extreme filth and neglect of treatment, and the irritation and course of the disease has tended to a fatal termination by pneumonia and hyperæmia of the brain.

Treatment.—Such applications are to be made to the skin that, while they tend to kill the *Acari*, they will not increase the irritation of the dermis; and if the *Acari* are thus destroyed, the eruptions will in general subside in due course.

If much irritation of the skin prevails, warm baths are to be prescribed, and opium may be given internally.

In healthy adults the whole body of the patient ought first of all to be thoroughly scrubbed over with good black (soft) soap, and the process continued for at least half an hour. The patient should then get into a warm bath, in which he should remain for another half hour. Having thus washed and dried himself thoroughly, he is to rub himself over with the following ointment:

R. Subcarbonatis Potassæ, ʒj; Sulphuris, ʒij; Axungiæ, ʒxij. Misce.

[The ointment used at the St. Louis Hospital, Paris, by Dr. Hardy—who revived the method introduced by Bourdin (1812) and materially abridged the time employed—is composed of sulphur ointment, to which subcarbonate of potash is added in the proportion of half a drachm to the ounce; 3 to 4 oz. of ointment, and 1 lb. of soap, are used to one patient. The ointment is made more certain by adding ʒj oleum anisi.]

Next morning a warm bath is to be taken, to clean the surface of the body from the remains of the anointing of the previous night. The cure ought now to be complete, so far as the destruction of the *Acari* are concerned (HARDY, HELMERICH, ANDERSON). The genuine pomade of Helmerich is one-third stronger than that which has been just quoted from Dr. Anderson, who considers it too irritant. The potash in the black soap and ointment acts as a solvent of the epidermis, and thus allows the sulphur to come into more immediate contact with the *Acarus*. A warm bath and plenty of hot water ablution completes the cleansing process.

The treatment may, however, be inapplicable to children, females, and men with delicate skins, or constitutional affections of the skin.

In them, although the principle of treatment is the same, the process of cure must be more slowly conducted by less powerfully

irritant substances. The patient having cleansed himself thoroughly in a warm bath, with ordinary yellow soap, the following lotion may be applied:

R. Calcis, 3ss.; Sulphuris, 3j; Aquæ, 3vij. These ingredients are to be boiled and stirred constantly till a homogeneous mixture is produced, which is to be strained through a sieve. [The fluid is to be decanted and kept in a well-stoppered bottle.]

These ingredients ought to produce a quantity more than sufficient for one person, and should be rubbed into the skin, not too roughly, [for half an hour], every night for several evenings. The cases of scabies in the Belgian army are treated by this lotion, (VELMINSKZ, ANDERSON), [KENDALL, NICHOLLS].

When the person affected is predisposed to *eczematous* eruptions, the following application is recommended:

R. Sulphuris, Olei Fagi, āā 3vj; Saponis Viridis, Axungiae, āā lbj; Cretæ, 3iv. Misce.

This ointment should be well rubbed in, after the skin has been prepared for it by the use of the warm bath and cleaning the body with common yellow soap. The potash in the black soap of the ointment acts as already stated, the chalk tends to remove the epidermis mechanically, the tar counteracts the tendency to eczema, and the sulphur destroys the *Acari*. The ointment ought to be left on overnight (if the skin is not too irritable), and should be washed off in the morning (WILKINSON, HEBRA, ANDERSON). Specific printed directions should be given to each patient; and cards are useful for this purpose, similar to those in use in the Dispensary for Skin Diseases in Glasgow. The following are the directions *printed* on each card, and which is given to each patient along with the quantity of ointment required:

"1st. Scrub the *whole* of your body (except the head) as firmly as possible, without hurting yourself, with black soap and water.

"2d. Sit in a hot bath for twenty minutes, or if you cannot get a bath, wash yourself with hot water thoroughly.

"3d. Rub some of the ointment thoroughly into the skin of the whole body (except the head) for twenty minutes. Let the ointment remain on the body all night.

"Repeat these processes every night for three nights, and then return to the dispensary.

"Besides, put all your washing clothes into boiling water, and iron all your other clothes *thoroughly* with a hot iron."

If such methods are systematically carried out, itch cases ought never to occupy hospital beds, either in civil or in military life.

The ordinary compound sulphur ointment of the pharmacopœia is also an efficient remedy.

[Dr. Pastau, of Breslau, has recommended *liquid storax* as the most certain of antipruritics, and it is said to have been successfully adopted in

the treatment of the disorder in the Prussian army. R. Styrae. liquid, 3j; Olei Olivæ, 3ij; M. One or two embrocations of f3ss. over the body, the head excepted, after a warm bath, are, it is claimed, sufficient. The oil of *petroleum* has been highly recommended by Dr. Decaisne, a Belgian army surgeon, who says it is almost an immediate cure for scabies, killing the *acarus* instantaneously, and is a disinfectant against the larvæ in the wearing apparel and bedclothes.]

The cure being complete so far as the person is concerned, care must be taken to destroy the *Acarus* and its eggs which may be amongst the clothes of the patient. For this purpose they should be exposed to hot air at the temperature of at least 150° Fahr., or, if possible, boiled in water, or exposed to the action of sulphur by steaming them amongst sulphur vapors.

[Living acari never wander, like pediculi, into clothing, and where there is a second contagion, it is from the ova in the detached crusts, which adhere to the clothes or bedding; but it would appear to be rare. In the General Hospital at Vienna, it is stated, that the clothes are never disinfected, and the relapses are only one per cent.; and persons often sleep in their dirty beds, after treatment, without recontracting the disease (GUDDEN).]

SECTION III.—EPIPHYTES—*Vegetable Structures; and the Pathology of the Lesions with which they are associated.*

These parasites are microscopic growths which belong to the lowest class of vegetable existence—namely, the *Fungi Cryptogamia*. Most of them are composed of simple sporules, germs, or of cells placed side to side or end to end.

Although much has been written on the influence of *fungi* in the production or aggravation of disease in the animal as well as in the vegetable kingdom, yet the subject is involved in much confusion, because in a few instances only have the persons recording their experience been sufficiently acquainted with the botanical nature of *fungi* in general, so as to give anything like a complete history of the cases which have fallen under their observation. Mere *mycelia* have been described as perfect plants, and mistakes have been made in important points of structure. Productions of an undoubted fungoid nature have been referred to *algæ*, although agreeing with them neither in habit nor in physiology, while the commonest moulds have received new names; and several conditions of the same species have been recorded as productions differing in their mechanism and physiological laws (BERKELEY). *Fungi* are the most numerous of all plants in regard to genera and species, and their growth is associated with most extensive injury to animal and vegetable life; and as they are now proved to be capable of propagation by implantation from animals to man, they demand from the physician a most careful study. The diseases of plants as well as animals have hitherto been almost neglected by the pathologist; yet how do we know that the *blights* of plants, or the causes of them, are

not communicable to animals and to man? It is known how intimately the diseases of man and animals are related with the occurrence of famines and the prevalence of unsound or unwholesome food, and of famines with the diseases of vegetable and animal life, as much as with the destruction and loss of food. The black sporules of *Ustilago hypodytes*, which cause disease in grasses in France (the grass smut), and those of *Ustilago vittata*, which cause similar disease in the grasses of India, are known to produce most injurious effects upon the haymakers in the former country (LEVEILLE). In places favorable to the multiplication of *fungi* they often commit extensive ravages. Among the silk-worms in the silk manufactories of Italy, *fungi* are the cause of more extensive destruction of such animals than we have any correct idea of. Under the names of "mildew," "blight," "smut," "brand," and the like, *fungi* commit extensive damage among living plants, as the farmer and orchardist know too well. It has been asserted that *fungi* are uncommon in tropical countries; but it is doubtful if this is true; and the *fungus disease of the foot in India*, so well described by the two Carters, show that *fungi* are capable of giving rise to a disease almost dangerous to life in that country. It therefore behooves the pathologist to study carefully the nature of those diseases in animals and vegetables, as well as in man, whenever he has an opportunity, and especially in India. It is not in all cases easy to determine whether they are the cause of morbid states, or whether, as some think more likely, the diseased tissue has merely afforded a suitable nidus for their development. It is certain that wherever the normal chemical processes of nutrition are impaired, and the incessant changes between solids and fluids slacken, then, if the part can furnish a proper soil, the cryptogamic parasites will appear. The soil they select is for the most part composed of epithelium or cuticle, acid mucus or exudation. Acidity, however, though favorable for their growth, is not indispensable; since some of the vegetable parasites grow upon alkaline or neutral ground, as on the ulcerations of the trachea, or in fluid in the ventricles of the brain. Certain atmospheric conditions seem favorable to the occurrence of those vegetable parasites. For example, *Tinea tonsurans* may be quite absent for years in places such as workhouses, where it commonly exists, and then for several months every second or third child in the place gets the disease. It has been observed that some of these parasitic diseases can be propagated by transference of the plant, as in various forms of *tinea*, and that the disease can be cured with the greatest readiness by the chemical agents which are most destructive to vegetable life.

These vegetable parasites have been shown to be capable of transmission from animals to man. It has been recently proved by Devergie that *T. tonsurans*, for example, is transmissible by contagion from horses and oxen to man, and that the parasite has given rise either to the same form of *tinea* disease or to another; but he ascribes both species of parasitic disease to the growth of one and the same parasite—namely, the *Trichophyton*. Von Bärensprung, of Berlin, bears similar evidence. He rubbed on his forearm some of

the scales of *linea* from one of the lower animals, containing abundance of the spores and mycelium of the fungus *Trichophyton*. No effect was produced for several days; but after a long interval considerable itching called his attention to the part, which he found occupied by a well-marked spot of *T. circinatus* about the size of a sixpence. In three weeks the patch increased to the extent of a crown-piece (*Brit. and For. Med.-Chir. Review*, July, 1857, p. 263).

Instances have been noticed of grooms being attacked by *T. circinatus* and *sycosis* after grooming horses affected by *T. tonsurans*. "A dragoon came to the Dispensary of the St. Louis Hospital affected with *T. circinatus* on the front of the right forearm. He stated that five or six of his comrades had contracted this affection as well as himself from grooming diseased horses. A visit to the barracks showed three horses with round patches absolutely identical with *T. tonsurans*. These were situated on the withers, shoulders, back, and belly. The hairs in the centre of each patch were broken off close to the skin, and there was a whitish, squamous, crust-like production, which was traversed by the hairs. The presence of sporules was detected by the microscope. The dragoon who showed the horses showed also his daughter, a girl of eight or ten years of age, the side of whose nose exhibited a patch of *T. circinatus*" (BAZIN, quoted by Dr. Anderson *On the Parasitic Affections of the Skin*, p. 51).

With regard to the transmission of *favus* from the lower animals, I am informed by Dr. Anderson that, in the Dispensary practice of Glasgow, the physicians often find it traceable to contagion from mice, cats, and dogs, similarly affected; but that mice are the animals which seem to be the ultimate source of the disease.

[Muller observed it in a Cochin-China fowl, and in several chickens which had contracted it from the fowl; Gerlach speaks of its transmission from fowls to the human subject; and Bazin relates an instance of the transmission of *favus* from an animal to the human subject: several mice caught in a trap were seen to have elevated, circular, and dull yellow-tinted crusts upon the head and forelegs; there was also a depression in the centre of each crust, and where it had fallen off the skin was ulcerated. The mice were given to a cat, which, some time afterwards, had a crust above the eye similar to those on the mice. Later still, two young children who played with the mice became affected with yellow crusts on the shoulder, face, and thigh, which were recognized as those of *favus*. Bazin examined some pieces of the crusts, and detected the parasite.* Dr. Anderson, who has recently paid much attention to the occurrence of vegetable parasitic diseases amongst the lower animals, and their transmission to man, mentions several authenticated cases which have come under his notice. (1.) A dog had a patch of *favus* upon one of his fore-paws, as proved by a microscopic examination of a portion of the crusts. This dog was in the habit of killing mice, some of which, on being caught, proved to be affected with *favus*, the *Achorion Schönleini* being found under the microscope. (2.) A woman and her child came to the Dispensary

* [*Leçons Théoriques et Cliniques sur les Affections Génériques de la Peau*, professées par le Dr. Bazin.]

sary with favus of the non-hairy parts of the body. The father and two other children were similarly affected. Mice previous to this had abounded in the house, and a cat was got to kill them. On examining the cat numerous favus cups were detected on the tops of its forepaws. (3.) An eruption was noticed on a little girl, and soon afterwards a sister, the mother, the baby, and a work-girl were similarly affected. The disease proved to be favus. Five weeks previously a number of mice had been caught in the house, and which had been much handled by the children. Several mice in the house were then caught and examined, and on the back of one of them, near the tail, a characteristic favus cup was seen, while the sides of the ears and head of another were eaten away by the disease. On the crusts being examined with the microscope, the *Achorion* was detected in great abundance.*]

On the other hand, it is shown that animals may contract parasitic diseases of the skin from human beings similarly diseased. Dr. Fox mentions an instance of a white cat, a great pet with the children of a family of nine, which contracted the *mange* and *T. tarsi* from *T. tonsurans* affecting five of the children. The fungus of the *mange* in the cat is the same fungus as that of *Tinea* in man—namely, the *Trichophyton*.

[Köbner succeeded in producing favus in rabbits, by inoculating them with the *Achorion* taken from the human subject.†]

The principal vegetable parasites associated in man with special morbid states have been enumerated as follows:

1. The *Trichophyton tonsurans* vel *Achorion Lebertii* (ROBIN), which is present in the three varieties of *Tinea tonsurans*—namely, *T. circinatus* (ringworm of the body), *T. tonsurans* (ringworm of the scalp), and *T. sycosis menti* (ringworm of the beard).

2. The *Trichophyton sporuloides* (VON WALTHER), together with the above, which are present in the disease known as *Plica* vel *Tinea polonica*.

3. The *Achorion Schönleinii* (REMAK) and the *Puccinia favi* (ARDSTEN), which are present in *Tinea favosa* (the honeycomb ringworm).

4. The *Microsporon mentagrophytes* (GRUBY), which is present in *Sycosis* or *Mentagra*.

5. The *Microsporon furfur* (EICHSTADT), which occurs in *Pityriasis* vel *Tinea versicolor*.

6. The *Microsporon Audouini* (GRUBY), which is present in *Porrigo* vel *Tinea decalvans* (*Alopecia areata*).

7. The *Mycetoma* vel *Chionyphe Carteri* (H. V. CARTER, BERKELEY), which gives rise to the disease known as “the fungus foot of India,”—a cotton fungus occurring in the deep tissues and bones of the hands and feet.

8. *Oidium albicans*, or “thrush fungus” (ROBIN) of diphtheritis and aphtha.

* [On the Non-identity of the Parasites met with in Favus, *Tinea tonsurans*, and *Pityriasis versicolor*. By Dr. MCCALL ANDERSON. *British and Foreign Med.-Chir. Review*, vol. xxxviii, 1866.]

† [Klinische und Experimentelle Mittheilungen aus der Dermatologie und Syphilidologie, von Dr. Heinrich Köbner. Erlangen, 1864.]

9. *Cryptococcus Cerevisæ* (KUTZING), *Torula Cerevisiæ* (TURPIN), yeast plant in bladder, stomach, &c.

10. *Sarcina ventriculi* (GOODSIR), or *Merismopædia ventriculi* (ROBIN), in the stomach.

It would have been better if these *fungi* had been described in the first instance without specific names. The fact of specific names having been assigned to each of them has drawn attention from the important part which these *fungi* perform in the work of decomposition. They may be forced to fructify by placing them in a globule of water surrounded by air, and placing them in a closed cell; and until the fungus has thus come to maturity it is worse than useless to give them names—so many different forms in an undeveloped state being all capable of reference to one common mould (BERKELEY).

The plants forming on mucous membranes, or in the contents of cavities lined by mucous or serous membrane, are in most cases only of secondary formations, and their exact pathological significance is unknown.

The pathognomonic sign of all the parasitic lesions of the surface in man and animals is the infiltration or destruction of hairs (*tinæ*) and epithelial textures (*muguet*, *thrush*, *öidium*) by the *sporules* of a fungus, and which, by union or by growth, form elongated branches, or *mycelium*.*

The diagnosis of such *fungi* on the skin, hair, or epithelium, can only be effected by a careful and skilful microscopic examination; and it is always absolutely necessary to use the *liquor potassæ* in the examination of all *tonsurant* appearances of the hair, of all *idiopathic* bald patches, and of all brown or yellow-colored scurfs, for *sporules* are frequently detected which had escaped observation before *liquor potassæ* had been used (BAZIN, FOX, ANDERSON). Sufficient time must also be allowed for the parts to become transparent under the action of this reagent.

The parasitic lesions of the skin are, as a rule, unsymmetrical, and hence they differ materially in this respect from syphilitic cases. They differ also no less essentially from the eruptive diseases of the skin. An eruption is no necessary part of these parasitic lesions; but, from the irritation established in the true skin, eruptions of various kinds may occur. Eruption thus often precedes the detection of a fungus, and, as a rule, very often follows its existence; and they who dispense with the microscope in the diagnosis of skin affections cannot avoid confounding severe eruptive with parasitic lesions, because they disregard the pathognomonic evidence of such lesions already indicated (FOX). The term *herpes*, therefore, as applied to these parasitic affections, is an objectionable term; because it has been already used to indicate a vesicular eruption, namely, *herpes zoster*, which invariably exhibits large typical vesicles.

There seems to be a peculiar condition of nutrition best fitted for parasitic growths of a vegetable nature, just as some constitu-

* These *fungi* have sometimes been popularly but erroneously termed *conferæ*. The *conferæ* belong to the *algæ*, and for the most part grow in salt or fresh water.

tions and states of the system are best suited for the propagation and development of entozoa; but the exact circumstances which predispose to the growth of these *entophytic fungi* upon the human body are not better known than those which predispose the body to receive and develop certain morbid poisons of a specific kind, known to multiply during the course of the disease, and to throw off material capable of propagating and spreading the same kind of disease. With regard to the parasitic diseases of the skin and hair, a failure of the vital powers to carry on the healthy processes of life seems ordinarily to be one of the inviting causes of such a development of true *fungi* as would constitute a disease. A special nidus or soil is necessary, just as a predisposition is necessary in the case of the spread of miasmatic diseases; yet care must be taken in both instances not to confound the co-operating cause with the special or peculiar poison or germ. Both Robin and Bazin recognize a condition of the hairs (dependent, perhaps, on constitutional causes) which appears to be essential for the growth of the plant; for sometimes the disease disappears spontaneously, and the fungus dies without treatment.

[Malnutrition and feeble health of the subject seem to favor the development of both vegetable and animal parasites,* and in scrofulous persons they are common and tenacious; but no condition is so fitting as uncleanness, the fungus there finding a good soil where it may rest unharmed, with no let to its growth, and this point is particularly insisted upon by Hebra. They affect particularly the skin of children.]

The evidence is now very strong which points to the various *fungi* already mentioned as mere varieties of two or more species (HEBRA, LOWE, HOGG, FOX, BERKELEY).

There are numerous facts which justify the belief that there exists but one essential fungus, whose sporules find a soil for development and growth upon the surface, or even within more secluded portions of the human body; and that varieties in the growth of that fungus are due to differences in the constitution of the individual, to the moisture, exudation, soil, or temperature under which the development of the fungus takes place. The exact nature of these differences is not yet understood; but the production of irritant acids and gases is a constant accompaniment of the growth of such parasites, by the chemical action of the vegetable cell; for it does not undergo development without exciting a chemical decomposition in the pabulum on which it feeds; and the different stages in its growth give rise to alcoholic, acid, and putrefactive fermentation. Of the latter there is ample evidence in many of these parasitic skin diseases, and especially in favus, the odor of which closely resembles that of some *methyamine* compound (LOWE). Thus their irritant action very soon may establish an eruption. But the ratio

* [Cl. Bernard states that frogs are subject to parasitic fungi, which in time cause their death; and that if a healthy frog is put into a jar with others so diseased, he will, for awhile at least, resist contagion; but if one which has been for some time in captivity, and whose nutrition is impaired, be thus placed, he becomes at once covered with fungous growths.]

of eruption to parasite is not constant; for an amount of fungus which will simply produce death of hair in one person or part of the body may in another produce irritation, eruption, or violent inflammation. These different results may be due to two causes,—(1.) Constitutional peculiarity in different individuals; (2.) Peculiarity of structure of a part as regards density, heat, moisture, and chemical and anatomical composition of the part. Not only do these fungus sporules penetrate hairs, hair follicles, and epidermic cells, but the development of sporules of a similar nature within the *bones* and deeper textures constitutes a most serious disease of the foot in India, about to be described.

It is not to be hastily concluded that a disease is non-parasitic because a fungus has not been demonstrated in the part where it was expected to exist. The minuteness of the sporules, and the care required in the investigation, sufficiently explain the very various opinions which have prevailed on the nature of these parasitic lesions (LOWE). To appreciate the ravages which may be produced by such minute cells, the reader may be reminded of the immense force which the growth of such cells is capable of producing through continuous and gradual development; for, being of so minute a form, and its agency so apparently invisible, its intrinsic power is apt to be underrated. An *Agaric* growing under a stone of more than a hundred pounds' weight will eventually raise it from its bed to the height of several inches; and the cells of that *Agaric* are not widely different from the *sporules* of a fungus. Again, the *mycelium* of a fungus (properly identical with that found in skin diseases) has been known to raise a cask of wine, the fungus feeding on the wine as it leaked from the cask (HARVEY, quoted by Lowe in *Lancet* of August 13, 1859).

The variations of form in these different vegetable parasites associated with the skin disease about to be described are obviously so slight that they seem insufficient to warrant different species being made out of them. They may all be initial or undeveloped forms, referable to the *Aspergillus glaucus* or *Penicillium*; the initial forms of both of these being isomorphous (LOWE); and both of them are also equally indifferent about the matrix where they grow, as long as the conditions for their growth are fulfilled (BERKELEY).

[In confirmation of this view Dr. Lowe states, that he placed in a bottle a solution of brown sugar and some favus matter, and exposed it to a moderately cool temperature for rather more than a month; at the end of which time the *Aspergillus glaucus* was detected in the solution, having, apparently, been developed from the favus matter; and this experiment was repeated several times with a like result.* Dr. Lowe admits the great difficulty of proof in such observations, from the minuteness of the objects under examination, and the impossibility of showing that no other plant of the same species was present to complicate results, myriads of spores of the same species always floating about in the atmos-

* [Transactions of the Botanical Society, vol. v, part iii, p. 193, quoted by Dr. McCall Anderson.]

phere, ready to rest and grow upon any suitable pabulum. Remak made like experiments, but was unable to reach any positive conclusion; while Köbner put the question to the practical test of repeatedly inoculating himself, Strube, and others with the *Penicillium glaucum*, without any effect. "Now," as Dr. Anderson very fairly remarks, "if the *Penicillium glaucum* were identical with the parasites of favus, ringworm, and Pityriasis versicolor, one would naturally have expected that he would have been as successful with it as he was in his inoculations with the *Achorion Schönleini*, the *Trichophyton*, and the *Microsporon furfur*;" and he thinks, "it must be conceded that further proof is required, before we can admit that the parasites productive of favus, Tinea tonsurans, and Pityriasis versicolor are identical with the *Aspergillus glaucus*" (ANDERSON, *loc. cit.*.)

Peculiarities have also been observed in the growth of the fungus, which may be explained as due to the stage of development and conditions of growth at the time it was examined (*Path. Society Trans.*, vol. vii, p. 395). The same fungi during their growth are known to assume very different forms and appearances. It thus happens that the same species has not only been described under different specific names, but even referred to different genera. Fries states that he has traced no fewer than eight genera of different authors to mere degenerations or imperfect states of one particular fungus (*Thelephora sulphurea*); and Nees von Esenbeck states that the same fungoid matter which develops a certain fungus in winter (the *Sclerotium mycetospora*) will develop another fungus in summer (the *Agaricus volvaceus*.) Professor Henslow showed that some of the supposed species of *Uredo* are forms of *Puccinia*, *Aegma*, and the like.

The identity of these fungi associated with skin disease has been likewise proved by clinical observation. In patients affected with ringworm of the head (*T. tonsurans*), patches of ringworm of the body (*T. circinatus*) are frequently seen on other parts of the skin, more especially on the neck, where the patches of the skin and scalp are often continuous (JENNER, ANDERSON). Instances of the converse of these observations are recorded by Dr. Fox, where the disease from the skin extended upwards to the scalp, with the characters of *T. circinatus* on the skin and *T. tonsurans* on the scalp (*Lancet*, Sept. 17, 1859). Hutchinson states that once, in examining with the microscope the parasite from a case of *T. tonsurans*, he inoculated himself by mistake, and there resulted a well-defined patch of *T. circinatus* on one side of the neck (*Med. Times and Gazette*, Jan. 12, 1861). Dr. Anderson mentions that in cases of *sycois* he has more than once found the external aspect or back of the hand or wrist the seat of *T. circinatus*, owing to the patient rubbing the itchy and diseased portions of the chin with these parts, thus giving an opportunity for the transmission of the parasite (*Parasitic Affections of the Skin*, p. 50). The ringworm of the skin, he also observed, might give rise to *sycois*. Dr. Fox relates that while examining a patient with *T. circinatus* he had two or three vesicles of *simple herpes* just appearing at the time on his own lip. The fungus became implanted amongst these vesicles; and the herpetic eruption

became irritable, inflamed, and pustular; ends were actually split up by the fungus way down to the follicle.

Sycosis is also shown to be produced from the implantation of the yeast plant; and *Achorion* have been seen in *T. tonsurans*. *Chloasma* may be produced from the implantation of the fungus of the "thrush" (Fox). The nail fungus is an *Oidium* (VIRCHOW), or an *Oidium* (KUCHENMEISTER); while the ear fungus is an *Aspergillus*. Figures used in illustration of the text (Figs. 86, 87, and 88, p. 88) show the sameness in the form of the diseases; and if contrasted with Fennell's admirable work on *The Principles of Dermatology* showing the sporules and mycelium of the fungi, it cannot fail to strike the observer that the fungi peculiar to the diseases about the hair are identical. Nevertheless, the lesions induced by the different fungi necessitate the description of separate diseases, for the treatment varies with the particular disease; and the clinical aspect of the disease is related to the stage of development, growth, and the fungus.

[The most eminent dermatologists are now of opinion that the pathogeny of the parasitic affections of the skin is to be denied. (1.) Some deny utterly the presence of fungi in these diseases, and have been described as such to be degenerations of the skin. (2.) Some admit that they are accidental and not essential formations. (3.) Some admit that parasitic fungi may be generated spontaneously on the skin where they are found. (4.) The camp is divided between those who believe that several fungi are the cause of the production of the parasitic affections, and those who maintain that they are due to the presence of one fungus. In a recent contribution to the *British and Foreign Medical Review* (July, 1866), Dr. McCALL ANDERSON, has treated the subject with great care, and who, in his *Parasitic Affections of the Skin*, was the first to recognize the correctness of Bazin's opinion—that *Tinea tonsurans* and *Tinea sycosis*, are all due to the presence of the same fungus, the *Trichophyton*—has presented very strong evidence of the correctness of the opinion. He has shown that the *Trichophyton* (the parasite of the three diseases) is the same as the *Achorion Schönleini* (the parasite of scald head), and the *Furfur* (the parasite of chloasma, Pityriasis, and other fungous growths). His proofs of the non-identity of the fungi are drawn from: (1.) The results of inoculation. (2.) The results of culture. (3.) Microscopic examination. The following evidence brought forward in support of his probability is that all cases of successful inoculation with the *Microsporon furfur*, the same parasitic dis-

that from which the parasite was taken. (2.) Of the innumerable cases occurring in the human subject, illustrative of the contagious nature of favus, *Tinea tonsurans*, and *Pityriasis versicolor* (*chloasma*) which have been recorded, there is no authentic case in which one of these diseases gave rise to one of the others. In one of Hebra's plates there is an instance of ringworm and favus on the same person at one time. This is no doubt very rare. Dr. Anderson says, he has never met with a case, and that amongst the 1300 cases of parasitic affections of the skin treated at the Dispensary for Skin Diseases, Glasgow, during the last four years, there were numerous examples of the contagious nature of tinea, favus, and *chloasma*, but there was not a single instance of one of these diseases giving rise, by contagion, to one of the others. He remarks, that when the coincidence happens, it is no more constitutive proof of the identity of these diseases than do instances of the coexistence of psoriasis and ringworm show the identity of these two affections. (3.) The difference in the appearance of favus, *Tinea tonsurans*, and *Pityriasis versicolor* (*chloasma*), when fully developed, is so very striking, as to lead to the belief that they are produced by separate parasites. (4.) There is no authentic instance on record of the transition of one of these diseases into one of the others. (5.) The difference in the appearance of the *Achorion*, *Trichophyton*, and *Microsporon furfur* is sufficiently striking to enable the observer in many cases to form a correct diagnosis from the microscopic examination alone.* (6.) Of the numerous instances on record of the transmission of favus and *Tinea tonsurans* from the lower animals by contagion or inoculation, favus has always given rise to favus, and *Tinea tonsurans* to *Tinea tonsurans*.]

RINGWORM—SYN., *TINEA TONSURANS*.

LATIN EQ., *Tinea tonsdens*; FRENCH EQ., *Tinea tonsurans*; GERMAN EQ., *Tinea tonsurans*—SYN., *Ringworm*; ITALIAN EQ., *Tinea tonsurans*.

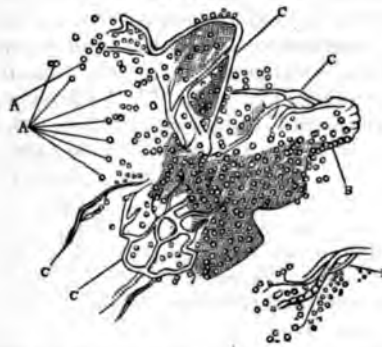
Definition.—An affection implicating the hairs of the skin, scalp, or chin, and usually assuming a circular form. The hairs become dry and brittle, having a tendency to crack or break across. Itching accompanies the primary eruption, which is generally at first erythematous, with slight swelling, and a fungus ultimately appears (*Trichophyton tonsurans*), which had been developing between the epidermis and the true skin. The fungus has a pure white color and powdery aspect. It covers the epidermis between the hairs, and forms around them a complete white sheath. Inflammation of the hair follicles and of the surrounding tissues occurs; and when pus forms, the fungus is destroyed at the expense of obliteration of the roots of the hair, when perfect baldness ensues (BAZIN, ANDERSON).

Pathology.—The nature of this disease is to be studied in the botany of the cryptogamic parasite called the *Trichophyton*, discovered by Malmsten in 1845. It consists of oval transparent spores

* [The spores of the *Achorion* are, on an average, about 3000th of an inch in diameter, and many of them are oval; those of the *Trichophyton*, are much smaller, being, on an average, about the 7000th of an inch in diameter; while the spores of the *Microsporon furfur*, although nearly as large as those of the *Achorion*, are more uniformly rounded, and have a remarkable and characteristic tendency to run together, and form clusters, like bunches of grapes (ANDERSON).]

or globules, about the $\frac{1}{1000}$ th part of an inch. Some of these are isolated; others constitute, by their union, isolated filaments. Comparatively few cryptogams possess a character which distinguishes the ringworm from other vegetable structures seen in the other parts of the body (Fig. 45). Its anatomical seat is in the hairs. The hairs and fungi simultaneously grow, and are larger than usual, are paler in color, lose their elasticity, break off, and some of them are found on the surface of the skin, so that the fungus is so that the surrounding skin is undisturbed. The hair breaks off from the epidermis, and the fungus fills the ducts, and the fungus is naked.

FIG. 45.*



which gives to the skin a rough anserine appearance, and the mycelium of the plants can sometime be seen as a white powder on the roots of the broken hairs. The hair becomes congested and thickened, and the fungus grows up with the scales of epidermis, with fatty matter, with pus and serous exudation, and so on. The thickness of the growth of the fungus varies in thickness in which the growth of the fungus exists in the *Herpes tonsurans* of Cazenave, the *scutulata* of Willan, the *Tinea tonsurans* of Erasmus Wilson and Dr. Brown. The only name called *Ringworm* is what is intended by those names now mentioned, and on which we agree.

There seems to be three varieties of the disease described under the following names:

1. *Ringworm of the Body* (*Tinea circinata*) is a rose-colored and slightly elevated spot, about the size of a piece, which shortly becomes the seat of inflammation, accompanied by tingling or itching. It gradually increases in size, but retains its circular form. As it tends, the healing process commences in the center, and the time the red spot is transformed into a latent ring, inclosing a portion of sound skin. For an indefinite period, the ring gradually increases, and have a diameter of four or five inches.

* Parasitic fungus from a case of *Tinea tonsurans*. Spores united at their ends; (c, c, c.) Empty tubes; (A, A, A.)

face affected is large, the circle is apt to become incomplete, so that various segments of circles appear. The disease may terminate spontaneously, the parasitic fungus being very superficial, the hairs small and rudimentary, so that the parasite dies for want of nourishment. It is apt to affect the face, the neck, the back, and outside of the wrist and hand (BAZIN, ANDERSON).

2. *Ringworm of the Beard* (*Tinea sycosis*) is met with on the upper lip and hairy parts of the cheeks, as well as on the chin, when it affects men; but the hairs of the axillæ or genital organs of females are not exempt from this disease. It commences exactly like *T. circinatus*; but it is not till the deeper structures are involved, and when small indurations occur, surmounted by pustules resembling *acne*, and when the hairs can be pulled out with ease, that the attention of the patient is attracted to the affection. The hairs are thickened, the bulbs flattened and more or less disorganized. The longitudinal fibres of the hair are separated by masses of sporules embedded between them; and where the fungus accumulates, nodes on the hair indicate the site of such accumulation. The medullary part of the hair is quite disorganized (Figs. 46 and 47), and may disappear altogether. In the advanced stage of *sycosis*, when inflammatory and suppurative phenomena prevail, the fungus is difficult to find.

3. *Ringworm of the Scalp* (*Tinea tonsurans*) is for the most part met with in children. It generally makes its appearance first in the form of rounded patches on different parts of the head, of a scaly or pityriasis-like inflammation. From the irritation induced small vesicles may form. The hairs in the first instance are dull, dry, twisted, and easily extracted; but as the disease advances they become very brittle, and break on attempting to extract them; and as they become more and more friable they break of themselves within a line or two of the skin (BAZIN, ANDERSON). The twisting of the hairs, so frequently observed, is due to—(1.) Plugging up of the follicular orifice by secretion, and detention of the upper part of the shaft of the hair, while its growth at the papilla still continues. Half an inch in length of hair may sometimes be pulled from beneath the false operculum. (2.) It may be due to the presence of *mycelium* in the follicle, clinging to the hair on one side and to the follicle on the other. It thus blocks up the follicle, and holds the hair (while still growing) to the diseased spot (Fox). The epidermis and stumps of the hair become covered over with a characteristic grayish-white powder, which ensheathes the hair. This powder consists of the *sporules* of the fungus. There is slight elevation and puffiness of the skin of the diseased parts, while its color is bluish or slate-colored in dark subjects, and grayish-red or yellow in fair persons (BAZIN, ANDERSON). This elevation and puffiness of the skin is due to a granular layer or *stroma*, which, on the addition of *liquor potassæ*, is seen under the microscope to be due to *sporules* of the fungus closely packed together. The amount of fluid influences materially the size of the *sporules* (ROBIN, Fox). When pustules and yellow crusts form, the detection of the fungus is more difficult.

The inflammation will last as long as the growth of *fungi* continues, but sometimes they die out spontaneously, and a perfect recovery takes place; but the hair follicles may be obliterated, when perma-

FIG. 46.*

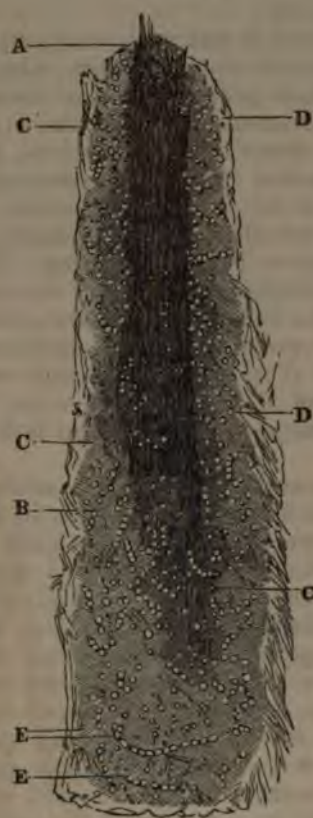
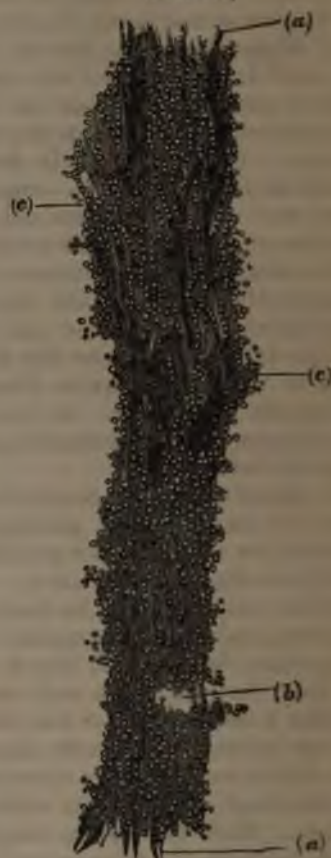


FIG. 47.†



nent alopecia of the affected parts is the result, with more or less atrophy of that portion of the scalp (BAZIN, ANDERSON).

The Treatment of ringworm, writes Dr. Parkes, has been long one of the most difficult points in dermatology. Its principles, however, are now well understood, and few cases resist the proper measures. The essential point is to apply to the roots of the hairs a preparation which may destroy the fungus: if this can be done, the disease

* Hair from a case of *Sycosis*.—(A.) Broken condition of the superior extremity of the hair; (B.) Rupture of the inferior extremity of the hair; (C, C, C.) Epidermic tunic of the hair; (D, D.) Isolated spores; (E, E.) Chains of spores (after BAZIN).

† Hair from a case of *Tinea tonsurans* loaded with *sporules*.—(a, a.) Broken ends of the hair; (b.) Rupture of the longitudinal fibres; (c, c.) Ragged edges of ruptured hair (Dr. T. M. ANDERSON).

is cured. It is first of all necessary to remove the hair. This is in part generally accomplished before the case comes under treatment, by the course of the disease; if it has not been sufficiently done, "epilation" can be accomplished by a chemical agent, or by extraction with pincers (Fig. 48). The forceps most suitable for this little operation are those about three inches long, having a weak spring, so that the hand may not be fatigued in using them. They should be made so that the two extremities come together very exactly, and do not slide the one upon the other. Each extremity should be a couple of lines broad, so that a fasciculus of hair may be caught up at one time when required; and should be furnished on the inside with very fine, but at the same time blunt, transverse denticulations, so that they may not cut across the brittle hairs. M. Bazin recommends an ointment composed of lime and carbonate of soda, of each one part, and thirty parts of lard, as an agent to remove the hair. The *oil of cade*, however, appears to be the best depilatory known, and with this mode of treatment epilation with the pincers may be combined. If the hairs are pulled out in the proper direction, there is very little pain, especially after the sensibility of the skin has been blunted by the use of the *oil of cade*. [After epilation, a lukewarm local douche should be used for ten or fifteen minutes.] The removal of the hairs permits a "parasiticide" solution to be applied to the hair follicles, within which are the prolific spores of the fungus. For this purpose M. Bazin recommends either a solution of *bichloride of mercury* (1 part to 250 of water) or an ointment of the *acetate of copper* (1 part to 500 of lard), about *two* grains to an ounce of water; and a little *alcohol* or *muriate of ammonia* should be used to facilitate the solution of the mineral. The *oil of cade* should be mixed with *glycerine* in the proportion of half a drachm to a drachm of the oil to an ounce of *glycerine*. Kuchenmeister's experience shows that the alcoholic solutions act most powerfully.

Dr. Parkes has used, with excellent effect, a solution of the *pernitrate of mercury*, about one part to thirty or forty of water. This is, however, a very powerful remedy, and is to be cautiously used, as it easily blisters the scalp; also an ointment composed of sulphate of copper (one part), alum (three parts), and lard (twenty to thirty parts, according to the age of the patient). Probably, however, a better parasiticide than any of these is the *sulphurous acid* [applied pure by means of a glass rod, or a piece of lint may be dipped in a saturated solution of the acid, and kept on the affected parts, with oiled silk over it, to prevent evapora-

FIG. 48.*



* Forceps for epilation (Dr. ANDERSON).

tion], as employed by Dr. Jenner, of University College, London, with astonishing results.

[Dr. Jenner also speaks highly of the results from the use of *sulphur ointment*, to an ounce of which forty grains of *hydrargyrum ammoniatum* is to be added. Dr. Laycock, of Edinburgh, recommends an alkaline lotion (Potassa Bicarb. $\mathfrak{z}\text{j}$; Aqua destil. $\mathfrak{f}\mathfrak{z}\text{iv}$), to be first used continuously for several days with the view of dissolving the crusts, and applied on lint by means of a mask of oiled silk, the hair being clipped to the level of the skin, and the parts fomented twice a day with warm water; and then brushing over the affected parts twice a day with Liq. Potassæ Arsenitis, $\mathfrak{f}\mathfrak{z}\text{ij}$; Glycerina, $\mathfrak{f}\mathfrak{z}\text{x}$. Dr. Masse extols a lotion of creasote, alcohol, and water.]

Dr. Bennett recommends the internal use of cod-liver oil at the same time. My friend Dr. Davidson has found the following method of treatment to succeed: namely, to apply *tincture of iodine* to the affected parts twice a day for fourteen days, and afterwards ointment of the *bichloride* of mercury (Corros. sublimat.). After the third or fourth application of the iodine, the disease will cease to spread, and the hair (which may have been thinning rapidly) will cease to fall off. A kind of crust is formed by the application of the iodine, which will scale off in the form of a scurf when the ointment is applied. Washing the head after the disease has commenced, and before medical treatment is begun, sometimes tends to spread the disease to parts of the head which had been sound before.

FAVUS—SYN., SCALD HEAD.

LATIN EQ., *Tinea favosa*—Idem valet, *Favus*; FRENCH EQ., *Tinea Favosa*; GERMAN EQ., *Tinea favosa*—Syn., *Favus*; ITALIAN EQ., *Tinea Favosa*.

Definition.—*A fungus parasitic disease, composed of cup-shaped scabs, sometimes distinct and separate, at other times indistinct or confluent. These fungi (Achorion Schönleini, Puccinia Favi) are capable of being implanted by transference from one person to another. The hairy scalp is its most common site, but the disease may be developed on the face, neck, or limbs.*

Pathology.—The disease has been found to depend on a cryptogamic fungus, which has been named the *Achorion Schönleini*, after Schönlein, who was the first to suggest that the *yellow favus* crusts in *Porriago lupinosa* and *P. scutulata* were constituted by a vegetable parasite. The disease with which it is associated is now called indifferently *Favus*, *Tinea favosa*, or *Porriago scutulata*. The primary seat of the parasite is in the depth of the hair follicle, outside the layer of the epithelium which covers the root of the hair, and which forms the "inner root sheath" of Kölliker. By using a concentrated solution of *liquor potassæ*, to make the parts transparent, the fungus may be observed with the microscope in the follicle round the hair at the place where it passes through the epidermis. A second form of the disease is that in which the plant is found in depressions on

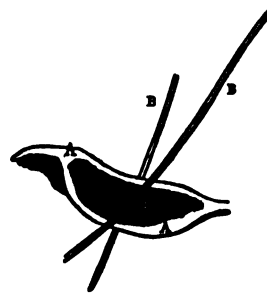
the surface of the skin, forming the yellow honeycomb-like masses which gave the specific name *favus* to the disease, and which, from their frequent buckler-like shape, suggested the term "scutulata." A cuticular elevation is seen, beneath which is a small favus. When the cuticle is raised, a drop of pus sometimes issues; hence the error of those who have considered this disease always pustular. Generally, however, there is no pus or liquid of any kind: the plant grows, and the cuticle over it (supposing it has not been forcibly detached) finally separates, leaving the favus exposed to the air. A third form of the disease is that in which the fungus attacks the nails, and occurs for the most part in those who have been long affected with the favus of the hair follicles, the fungus taking root and germinating beneath the nail (Fig. 49). After the spores have commenced to germinate between the superficial and deep epidermic layers, the nail becomes thickened over the affected part, and its color becomes gradually more and more yellow, owing to the favus matter shining through it. As the fungus increases in growth, it gradually presses on the nail, rendering its longitudinal striæ very evident, and ultimately leading to the formation of fissures in it. As the pressure of the nail increases, its substance gets thinner and thinner, till perforation occurs; and then a favus cup makes its appearance externally, but more or less deformed (ANDERSON).

FIG. 49.*



It is important to notice that at first there is, at the point where the favus is about to form, only an increased secretion of epidermis; and sometimes the under surface of the favus is coated by cuticle, which separates it from the compressed and attenuated derma. As it increases in size, and becomes more prominent, the epidermic covering is ruptured. Each favus crust is also enveloped in a capsule of amorphous structure, within which is inclosed the true favus matter (Fig. 50).

FIG. 50.†



The favus consists of the *mycelium*, the *spores*, and the *receptacles* of the *Achorion*, together with a finely granular amorphous layer, which forms the external coat of the favus, and is the representative of the amorphous "stroma" which often accompanies the mycelium of *fungi*. In the favus another and distinct fungus can sometimes be found—namely, the *Puccinia favi*—which is easily recognized: it has one extremity (the body) rounded and composed of two cells

* (A, A.) Upper surface of nail; (B, B.) Lower surface of nail; (C, C.) Favus matter (white in the wood-cut, yellow in the original), running upwards and forwards between the laminae of the nail (ANDERSON).

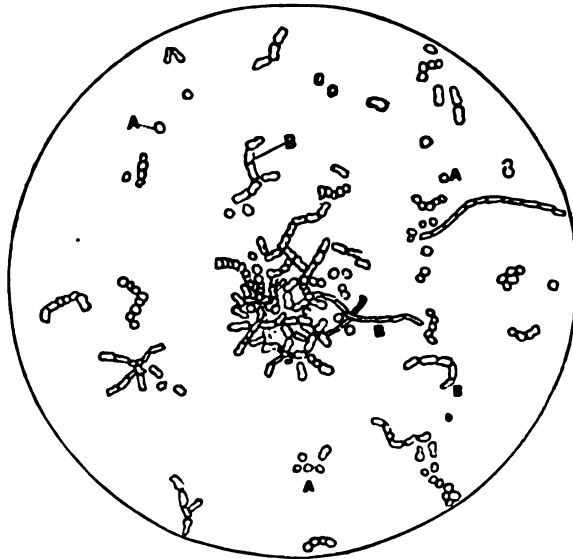
† Favus cup.—(A, A.) Amorphous envelope; (C, C.) Favus matter; (B, B.) Hairs traversing the Favus cup (ROBIN).

of unequal size, a superior and an inferior; the other extremity is prolonged into a jointed stem or trunk.

When a little of the favus matter is broken up and examined microscopically, after being acted upon by solution of potash, it is seen to consist of numerous little oval or rounded bodies, the sporules of the fungus having a diameter of about $\frac{1}{1000}$ th part of an inch (Fig. 51).

A number of cells united end to end form simple or jointed and branching tubes (Figs. 52 and 53), developed from the sporules (Fig. 51). Little granules or nuclei may be seen in the interior of

FIG. 51.*



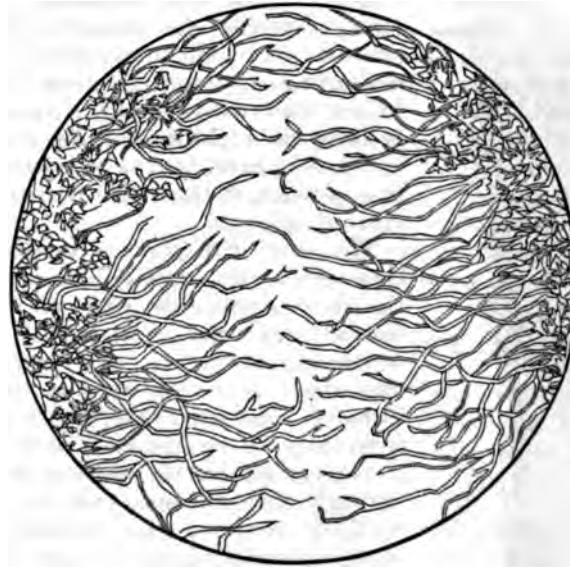
the spores. The tubes vary in diameter, and hairs in the vicinity of the favus crusts are impregnated with the fungus. The disease has been communicated by inoculation from man to man, and from mice to cats, and thence to man (BAZIN, DRAPER, FOX, ANDERSON).

Symptoms.—Favus is the most common and the most inveterate form of scald head. The disease commences with a slight pruritus or itching of a few hours' duration, followed by an eruption of small red *vari*, sensible to the touch and to the sight. These augment in size, and, before twelve hours have passed, a yellowish point forms on each of their apices, at first so small as to be only visible under a glass of considerable power. The surface appears now as if covered with specks of a sulphur-yellow color, and each *varus* appears as if set in the skin, with an umbilicated or de-

* Fungus matter from a favus crust.—(A, A, A.) The isolated sporules; (B, B, B.) Chains of sporules (Dr. ANDERSON).

pressed centre. If any fluid exudation exist, it does not remain so, but concretes into a dry, brittle, candied, honeycombed-looking

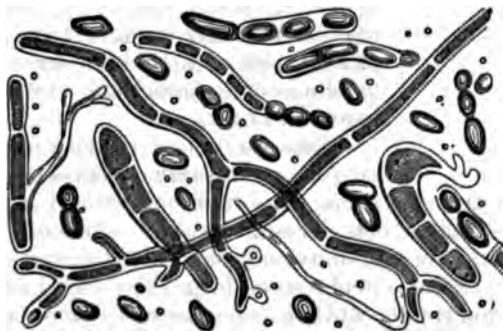
FIG. 52.*



$\frac{1}{1000}$ th of an inch \times 840 diameters (Dr. ANDERSON).

scab or crust, which retains the form of the pustule, is similarly cupped or depressed in the centre, covered by the epidermis, while

FIG. 53.†



* Fungus matter from a favus crust, showing branching tubes running inwards to the centre of the figure from the epithelial scabs and sporules at the edges (ANDERSON).

† Showing the mode of reproduction of the *Achorion*, or fungus of the favus (after BENNETT).

its under surface is marked by a small mammary process which corresponds to the depression of the pustule. The honeycombed

FIG. 54.*



appearance of the scab gives the peculiar character of the disease, and hence the term "favus." The crust continues to increase, still preserving its circular form and depressed centre, till it occasionally attains a magnitude of five to six lines in diameter. When the crust is recent, it is of a yellow or fawn color; as it becomes older its hue becomes lighter, and, as it is easily reduced to a powder, it has been compared to pulverized sulphur.

The number of favi is considerable, and they commonly appear in crops, affecting the same or different parts of the head at distant intervals. They may be either distinct or confluent. When very numerous, they are confluent, but the cupped form of the individual crusts may still frequently be recognized; and according to Rayer, should this peculiar form be lost through the copiousness of the secretion, still, by removing the superficial layers, each particular favus, with its central depression, may in general be made out. At a more advanced stage of the disease the epidermis disappears, and a viscid fluid is secreted in such abundance as to form one entire incrustation over the whole head; hence the *Porriago larvalis*—mask or vizor-like scald head. The smell of the scab is peculiar, and has been compared to that of the urine of a cat, or of a cage in which mice have been kept. It is probably due to a species of alcoholic fermentation (*methyamine*) in connection with the vegetable growth (LOWE).

When a crust of recent formation is removed, a circular depression, wider and deeper than the favus, is seen. At a more advanced stage the ulceration penetrates below the dermoid tissue. Indeed, Alibert says he has never been able to remove a crust, for the purpose of making a preparation, without deeply wounding the scalp, and producing considerable hemorrhage, while in some cases a deep and extensive ulceration takes place, which has penetrated even to the bones of the cranium.

* Hair with favus fungus.—(A, A.) Chains of sporules projecting beyond the edges of the hair; (B.) Sporules between the fibres of the hair; (C, D.) Broken-up root end of the hair, with masses of sporules between the lamina (after KUCHENMEISTER).

The *Porriago lupinosa* and *Porriago scutulata* are accidental varieties, in which the scab resembles a lupine, rather than the cell of the honeycomb, and is very rarely seen; or the appearance of the scab is shield-like; and when of some extent and well marked, the patch is soft, doughy, and painful when pressed upon. Some of the hair appears to be removed by the roots, while other portions are broken off near the scalp, the roots remaining. Those which remain are readily removed by friction, and if pulled, have scarcely any hold of the scalp.

Treatment.—The treatment of the various forms of *favus* is now very strictly determined. Some practitioners, however, still rely entirely on a constitutional treatment, such as small doses of rhubarb and soda, small doses of mercury, some preparation of iron; or on vegetable tonics, as the infusion of cascarrilla or compound infusion of gentian. With such treatment, if the health improves, it is believed the fungus will spontaneously disappear. Others, again, as entirely rely on a local treatment, attempting to exterminate the disease by cauterization, or by applying some favorite ointment; and the catalogue of ointments used for this purpose includes all that have at any time been admitted into the pharmacopœia.

As in the last-described disease, the cryptogamous parasite must be destroyed, and its germs eradicated.

The best method to accomplish this, is, in the first instance, to shave the head and apply a poultice till all the scabs, or nearly so, are removed; and this being effected, the whole hairy scalp, or site of the *favus* fungus, should be anointed with some of the following applications: The *tar ointment* (*ungt. picis liquidæ*) has hitherto been the orthodox application. This ointment should be washed off night and morning with soft soap and water, and be as often reapplied. The head should be shaved twice or thrice a week, and where there are other children, the affected child should be isolated as much as possible, to prevent the disease from spreading. This form of *porriago*, in the early stages, will sometimes yield by washing the part with the *oleum terebinthinæ* night and morning, and cutting the hair close.

The *Favus scutulata* is a disease often rebellious to every mode of treatment, but, applied at a favorable moment, a simple method may succeed. Dr. Willis has seen the disease yield to fomentations, or to bread poultices. The application of the lunar caustic round the patches, about a line from their outer margin, is another favorite method of treatment. In the latter periods of the disease, Dr. Willis recommends—

A solution of *Sulphate of Copper*, in the proportion of seven grains to ten ounces of water; or of the *Nitrate of Silver* in the same proportions. The mild ointment of the *Nitrate of Mercury*, a salve of the black sulphuret of the same metal (*Sulphuretum Hydrargyri Nigr.*, 3j ad 3ij, Adipis 3j); the *Unguentum Picis*, an unguent of the *Cocculus Indicus* (3j to 3ij, Adipis 3j), may be tried one after the other; and in different instances each will have the merit of the cure.

[Hebra, following the recommendation of Kuchenmeister, has succeeded with, first rubbing the parts with lint soaked in a lotion of *Vera-*

trin gr. v, *Alcohol* (80°) ℥j, and then laying over them a dossil wet with it. *Alcohol* alone will often effectually destroy the fungus.]

The most effectual remedy is unquestionably the eradication of the affected hairs, and the use of such parasitocides as have been already mentioned under the different forms of *Tinea*. Dr. Anderson finds that when *favus* affects the head, all treatment is absolutely useless except *epilation*. The other methods are merely palliative—the disease reappearing whenever the treatment is stopped. Dr. Bennet, of Edinburgh, places great faith in the olive oil treatment. It is very useful in many cases before proceeding to *epilation*. After the hair is cut short, and the *favus* crusts removed with poultices, the parts affected should be smeared thoroughly night and morning with fresh almond oil; and once or twice a week the head should be washed thoroughly with soft soap and warm water. After a few weeks of this treatment the hair becomes less friable, and *epilation* is much more easily and efficiently performed, and does not cause nearly so much local irritation. The hairs are to be removed *singly* with the forceps, not pulled out along with all the healthy growth in their neighborhood, as used formerly to be done by the barbarous application of the pitch-cap.

This disease occurring on surfaces not particularly covered with hair yields at once to the application of a solution of sulphate of copper, or of the nitrate of silver in water, or to the solution of sulphurous acid as recommended by Dr. Jenner. The treatment of *favus* recommended by Robin and Bazin is *epilation*, and the application of the corrosive sublimate solution, or of acetate of copper ointment (1 part to 500 of lard), to kill the plant still remaining adherent to the hair follicle.

BALDNESS IN PATCHES—SYN., *TINEA DECALVANS*.

LATIN Eq., *Tinea decalvans*; FRENCH Eq., *Tinea decalvans*; GERMAN Eq., *Tinea decalvans*—SYN., *Alopecia areata*; ITALIAN Eq., *Tinea decalvans*.

Definition.—A fungus disease, causing the formation of rounded or oval patches of baldness, sometimes solitary, more generally multiple. It affects the hairy scalp principally; but the beard, the genital organs, and hairy portions of the skin, may also suffer.

Pathology.—The fungus to which such circumscribed patches of baldness are owing has been named the *Microsporon Audouini*, detected by Gruby in 1843. It is present in the disease commonly called, after Willan, *Porrigio decalvans*, or *Alopecia circumscripta*; or, after Bateman, *Tinea decalvans*; by Bazin, *Tinea achromatosa*; by Anderson, *Alopecia*. It differs from the *Trichophyton* of *Tinea tonsdens* by its numerous waved filaments, and by the extremely small size of its sporules. It is not found, like the *Trichophyton*, in the interior of the root, but forms round each hair a little tube; the hair then becomes opaque, softens, and breaks off (Fig. 55). The *alopecia* is rapid, with or without previous *vittiligo* of the skin; the dermis is not congested, and the epidermis is thin and smooth.

In the early stage of the disease the hairs appear dull and lustreless, and more easily extracted than healthy hairs. The skin is reddened, swollen, and slightly itchy. A whitish matter may be seen on the diseased skin and hairs, which is due to the sporules of the fungus. The hairs suddenly fall off from the affected parts, and a round bald patch is left, which is perfectly white, contrasting in its whiteness with the parts of the scalp or skin provided with hairs. The fungus may also be developed in the nail, like favus (BAZIN).

There is an affection which should be distinguished from the *Porriago decalvans* (or *Alopecia circumscripta*), and which is characterized by a rapid disappearance of pigment from both skin and hair, with or without *alopecia*. M. Bazin includes it in his *Tinea achromatosa* (*Teigne achromateuse*), but does not mention the fact that *alopecia* is not constant; and states that a parasitic plant is present. It is probable, however, that something more than a fungus exists, to cause the total disappearance of pigment from a considerable portion of dermis. Besides, when the hairs return, they are at first quite white and downy, like those on children, and only gradually regain color; whereas, if the *vitiligo* were owing to a plant, they would most likely not grow at all. The disease appears to be allied to those obscure pigmentary changes which have a much deeper seat than the surface of the body (PARKES). *Vitiligo* is sometimes a congenital affection, and seems to consist in an abnormal distribution of the pigment of the skin; so that there are irregular patches which are quite white, and altogether wanting in pigment, but are surrounded by skin provided with an excess of coloring matter.

FIG. 55.*



* Fungus of the hairs resulting in *Alopecia* —(A, F.) Lower part of the hair; (F, G.) Root of the hair without the capsule; (C.) Spheroidal swelling of the hair, due to the accumulation of sporules; (E.) Between the longitudinal fibres of the hair; (D.) Rupture of the longitudinal fibres; (I.) Sporules and tubes of the parasite; (H.) A group of sporules proceeding from G, the ruptured root (ANDERSON).

The hairs proceeding from the portion of the skin deprived of pigment are similarly colorless.

The disease is not quite so readily transmitted as *Tinea tonsurans*, but still it is capable of being transmitted from one person to another, so that children so affected should be separated from their companions (ANDERSON).

Treatment.—This consists—(1.) In preventing the spread of the disease circumferentially. All the hairs, therefore, within a quarter of an inch of the circumference of the patch ought to be carefully extracted. The head should be washed daily with soft or black soap. All the *downy hairs* within the patch must be similarly removed till healthy hairs begin to grow; and some of the parasiticide lotions or ointments must be industriously used. (2.) Stimulants, or even blisters, must be applied to the surface of the bald patch after the fungus has been destroyed. A mixture of equal parts of *collodium* and of *ether cantharidialis* (*collodium vesicans*) is the most useful stimulant. The following lotion may be found advantageous to use alternately with the *collodium* stimulant, namely:

R. Liquor Ammoniaë, ʒiiss.; Ol. Olivæ, ʒij; Ol. Macidis, ʒss.; Spiritus Rosmarini, ʒiv; Aq. Rosæ, ʒij; *misce bene*. To be used night and morning applied over the bald patches.

TINEA [PITYRIASIS] VERSICOLOR—LIVER-COLORED SPOTS, OR CHLOASMA.

Definition.—A fungus affection of the skin, characterized by one or more broad irregularly shaped patches of a yellow or yellowish-brown color, occurring most frequently on the front of the neck, breast, abdomen, and groins, having a predilection for those parts of the body covered by clothing. The patches do not generally rise above the surface of the skin; and there is usually some degree of itching.

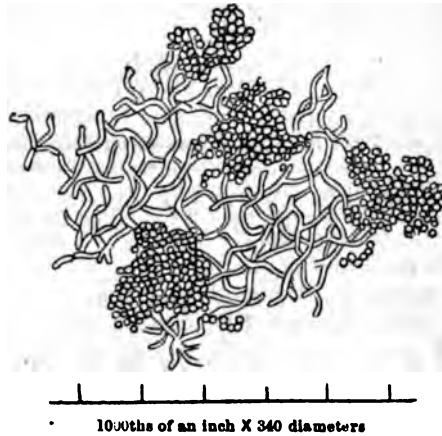
Pathology.—On passing the hand gently over the diseased surface, it may be found to be less smooth than the surrounding skin. It may be seen to be the seat also of a very fine desquamation, or at least of an abnormal condition of the epidermis. Thus far the surface of the affected parts may have a *dusty-like* appearance, like bran, and so may merit the name of *Pityriasis versicolor*; but in no other respect has it anything in common with ordinary *pityriasis*—a disease altogether unconnected with parasitic fungi. The scales which desquamate from chloasma have a yellowish color when contrasted with the white scales of such scaly diseases as *Pityriasis vulgaris* or *Psoriasis*. Hence the term Chloasma—from *χλωος*, “a greenish-yellow color”—appears more suitable than any of the other names by which the disease has been described.

The disease commences by little spots about the size of a pin-head, which tend to extend circumferentially; circular spots form and unite so as to produce large irregular patches, which may extend till the greater portion of the skin of the trunk is affected. The skin of the diseased parts has a peculiar brownish color, but the depth of tint varies from the slightest increase of color to a

shade almost black. The color has been said to resemble diluted bile.

A microscopic fungus, to which the name of *Microsporon furfur* was given by Robin, is the essential cause of the disease. It was discovered by Eichstädt in 1846. Soon afterwards it was described by Sluyter and by Sprengler, who gave a drawing of it. On putting a little of the dust from the desquamating surface under the field of the microscope, and adding a drop of *liquor potassæ*, scales of epidermis are seen mingled with the sporules and tubules of the fungus. The sporules are oval or rounded, and usually collected into large clusters like bunches of grapes, and are so characteristic as almost to be pathognomonic (ANDERSON). The tubes are short and branching.

FIG. 56.*



Dr. Anderson and Mr. Startin give numerous instances which prove that chloasma is a disease capable of being propagated from one person to another. It is a common affection with scrofulous persons especially, and may not unlikely be favored by wearing the same flannel day and night, neglecting to wash the body for fear of catching cold (ANDERSON). It is not uncommon for such people to wear the same flannel next to the skin for a week, a fortnight, three weeks, and among the poor even for a month. And it is by no means an uncommon thing for them to wear the same flannel night and day, not once removing it from the moment it is put on till the time it is considered desirable to have it washed. The consequences of such habits are an accumulation on the surface of the skin of its secretion, and of undetached epithelium, and the consequent formation of a nidus favorable to the growth of the *Microsporon furfurans* (Dr. Jenner, *Med. Times and Gazette*, 1857, p. 651).

Treatment.—Local applications constitute the principal part of the treatment. A solution of bichloride of mercury, in the proportion of two grains to an ounce of water, applied over the affected parts once or twice daily, is generally effectual in destroying the progress of the fungus. Mercurial or sulphur baths have a similar effect, either singly or combined, care being taken to avoid salivation. The use of black soap night and morning is recommended by Dr. Anderson, or the use of the following mixture:

* Shows the grape-like arrangement of the sporules and the short branching tubes of the *Microsporon furfur* in chloasma (after Dr. ANDERSON).

R. Bichloridi Hydrarg, ʒj; Alcoholis, ʒss.; Saponis Viridis et Aquæ destillatæ, aa ʒiiss.; Ol. Lavandulæ, ʒj; misce. To be used night and morning, in the same way as the black soap; but if the gums get tender, its use must be suspended.

Great attention must be paid to cleanliness, and the patient should change flannel clothes very often, and should not sleep in them, or at least not in the same flannels that are worn during the day.

MYCETOMA—Syn., MADURA FOOT.

LATIN Eq., *Mycetoma*—Idem valet, *Pes Madurensis*; FRENCH Eq., *Mycetoma*; GERMAN Eq., *Mycetoma*—Syn., *Madurafuss*; ITALIAN Eq., *Mycetoma*.

Definition.—*A disease due to the presence of a mucedinous fungus, which eats its way into the bones of the tarsus, metatarsus, and lower ends of the tibia and fibula. In process of time it tends to cause death from exhaustion* (CARTER, BERKELEY).

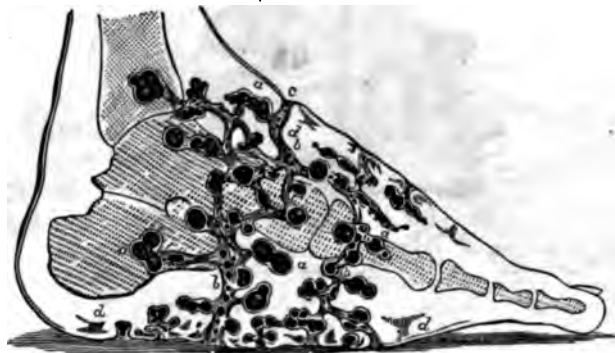
Pathology and Historical Notice.—Dr. H. V. Carter, the Professor of Anatomy and Physiology at the Grand Medical College of Bombay, made a report in March, 1860, on this formidable fungus disease. It occurs in many parts of India and the northeastern shores of the Persian Gulf. In the Bombay Presidency it has been seen at Kutch, Kattiawar, Guzerat, Scinde, the Deccan, and Lower Koncan. On the Madras side it has been seen at Gunttoor, Bellary, Madura, Cuddapak, some parts of Mysore, and at Trichinopoly. In the Bengal Presidency it prevails to a limited extent round Sirsa; but patients come from Bikaner, Bhawalpore, and Hissar. It is known amongst the Indian medical men as the "fungus foot" or "fungus disease of India;" or under the scientific names of *Podetoma* or *Mycetoma*, and by several characteristic native names. It is a disease which has hitherto occurred among natives only, and is undoubtedly due to the presence of a *mucedinous fungus*, which eats its way into the bones of the foot and lower ends of the tibia and fibula, penetrating or tunnelling through the tissues of the entire foot by numerous fistulous canals, tending to cause death by exhaustion, unless a timely amputation is made above the diseased part. The history of this disease is now rendered still more interesting from the fact that the Rev. M. J. Berkeley, M.A., F.L.S. (whose authority on *Cryptogamia*, and especially *fungology*, is well known), has succeeded in developing a peculiar mould—the perfect condition of the species—from the black fungous masses sent to him by Dr. Carter. The nature of the disease has thus been more clearly determined; and the account here given is taken chiefly from the description of the disease by Mr. Berkeley, in the second volume of the *Intellectual Observer*, p. 248, and from the writings of Dr. Carter, in the *Bombay Medical and Physical Society's Transactions*, which he kindly sent to the author.*

* [See, also, "On Mycetoma, by H. V. Carter, M.D., Lond., Assistant-Surgeon, Bombay Army." *Brit. and For. Med.-Chir. Review*, vol. xxxii, 1863.]

The fungus disease and material of the fungus assumes various forms, three of which may be considered typical:

1. The first form is that in which the bones of the foot and the lower ends of the leg-bones, just above the ankle (for the disease never ascends higher), are perforated in every direction with roundish cavities, varying in size from that of a pea to that of a nut or pistol-bullet (Fig. 57), the cavities being filled up with a dense fungous mass, of a sienna red within, but externally black, and resembling a small dark surface, from which a purulent fetid discharge is poured out, often accompanied by little pieces of the fungus. The masses and granules are embedded in a whitish semi-opaque glairy substance of homogeneous consistence, while the walls of the canals have an opaque yellow tint, and are readily torn. The whole of the surrounding softer parts are converted into a gelatiniform substance, taking the place of muscles, the tendinous and fatty

FIG. 57.*



structures being less readily changed. The foot presents externally the peculiar turgid appearance which it so often assumes in bad cases of scrofula. Besides the canals, pink stains or streaks are ob-

* The figure represents the general appearance on section of the diseased foot in the fungus disease of India. It is based upon dissections, and on three sketches made immediately after amputations of the limbs (CARTER).

(a, a.) The *fungi*, some of which are globular and of large size, others smaller and more irregular, and others mere granules. The former are lodged in the spherical cavities in the bones. (b, b.) The *canals* in the soft parts and bones which lead to the free surface of the skin. They frequently communicate, and are lined by a continuous membrane; in them are contained the *fungi*. (In a diagram of this sort it is impossible to represent the soft glairy material which also occupies the canals.) (c, c.) The *apertures* on the surface where the canals terminate. They are often very numerous, and frequently in them may be seen impacted the black particles. (d, d.) The pink-colored *stains* or *streaks* in the skin, above described. They are common to both varieties of the disease, and by them it is supposed the growth is multiplied.

It is to this variety of the affection that the term "fungus disease," which correctly expresses its nature, was, *par excellence*, originally applied. Hitherto no other instances of it have been distinguished, except those described by Dr. Carter; hence it may perhaps be regarded as comparatively unfrequent. The fungus particles or masses are of a deep black color, and of firm consistence; they are sometimes as large and as round as a pistol-bullet.

servable on the skin, and penetrating the subjacent tissues, filled with spherical or ovate groups of minute, bright orange-colored particles, and containing occasionally a few larger cells, the nature of which has not at present been ascertained, though it is conjectured that they present the earliest appearance assumed by new attacks of the disease. (Specimens of the disease are to be seen in the Museum of the Army Medical School at Netley.)

Of the structure of these large truffle-like bodies, the figure (58) copied from the *Intellectual Observer*, from a specimen examined immediately after amputation, will give the reader an idea. The parts in which the structure is most visible present precisely the characters of a true *Oidium*, such as *O. fulvum*. Short, beaded, tawny threads arise from a common base, consisting of cylindrical

FIG. 58.*



articulated filaments, having at their tips large spore-like cells. These, however, do not appear to germinate *in situ*, but to become enormously dilated, their albuminous contents assuming at length a resinous consistence, while many of them burst, and nothing remains except fragments of the old cell-walls. The resinous matter is inflammable, but its exact chemical nature has not yet been ascertained.

The fungus of the foot resembles closely the genus *Mucor*, but there is no *columella* in the *sporangium*—a character which accords with *Chionyphe* rather than with *Mucor*. Indeed, there does not seem to be a single character in which the fungus of the Indian disease differs generically from the *Chionyphe*. Its mature form is seen to be composed of a thin filamentous stratum, spreading in every direction over paste, on which it may be propagated so as to form little slightly raised patches.

* Structure of the truffle-like bodies, presenting the characters of *Oidium fulvum*,—short, beaded threads arising from a common base, consisting of articulated filaments, having at their tips large spore-like cells.

The species has been named by Mr. Berkeley as *Chionyphe Carteri*, the name serving to record the labors of the two Carters "united in their love of science, though not in consanguinity." It is highly probable, as Mr. Berkeley observes, that many of our common moulds occasionally commence with a similar condition. The first indications of vegetation on tainted meat or paste assume the form of little gelatinous spots, of various colors, consisting of extremely minute distinct cells, and these seem to be an early stage of a common species of *Aspergillus* and *Penicillium*, or other genera. If there be any truth in the notion entertained by Mr. Berkeley, that hospital gangrene depends upon some vegetation of this nature, acting as a putrefactive ferment, there may be good reason for believing that the red spots in question are really the commencement of the disease under consideration.

In the second form under which the disease appears the black fungous masses are entirely wanting, and in their stead masses are found of what looks like sloughing tissue. White granules, however, occur in the cavities and in the discharge, which appear to be a form of the same fungus, though the identity has not been proved. Under the microscope it wears the appearance of a congeries of large cells filled with smaller ones. Whether the perfect form of the plant be the same or not, the phases of the disease produced by it are exactly the same, and the malady admits of no other remedy than amputation of the foot.*

A third form of the disease is known under the name of the *Madura foot*, from its having occurred at Madura. In this case the foot becomes enormously enlarged about the instep, though not so much at the ankle, while the toes are hypertrophied, and almost lost or embedded in the mass. The small bones are nearly destroyed, leaving behind a pallid or reddish tissue, while the others are more or less excavated. There are the same canals and external sanious apertures. In some parts they are filled with the same fleshy tissue, in others lined with it, where large cavities are formed by the junction of several canals containing broken-up osseous tissue from the exposed bones around, gray fragments, and masses of pigment. The pink color is partly owing to a general diffusion of pigment, which tinges the oil-globules, and partly to the presence of very numerous single or aggregated elliptic particles. These granules are from the $\frac{1}{80}$ th to the $\frac{1}{10}$ th of an inch in diameter, and occur sometimes as single ellipses, sometimes as two combined at the extremities of their major axis, and sometimes as square bodies

* In the *second variety of Mycetoma* we find three or four different kinds of particles: these, however, are always light-colored and soft, and generally very small or minute. Of them only one form, certainly the more common, has been noticed by writers: it is that in which each particle is seen to be invested by a crystalline coat. The truly fungus nature of the more common kinds of granules or particles, and of that striking instance of the disease from Madura, is as yet only matter of inference.

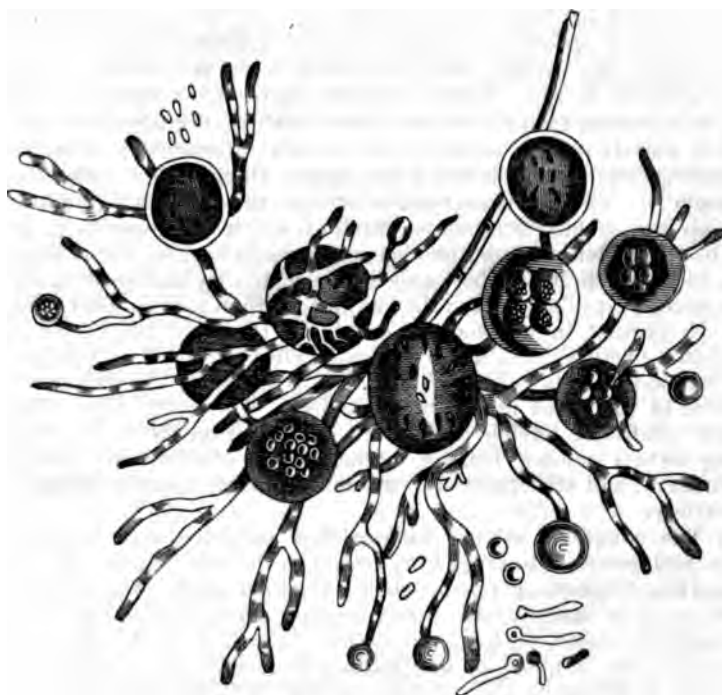
It seems desirable that every step in the investigation of this disease (the elucidation of which is committed, as it were, to the medical officers of India) should be based on direct and repeated observation, and that speculation be refrained from as being at least useless.

with rounded extremities, divided into four. They are quite visible to the naked eye, insomuch that, when the sawn surface is first exposed to view, it appears as if strewed with grains of red pepper; and pains were therefore taken by Dr. Carter to assure himself that they were not particles accidentally introduced through the open window. Further examination convinced him that, though different in color, they were similar in essence to the granules described in the second form. None of the black fungous masses appeared, but there were globular opaque bodies of various sizes, which now require notice, and which, though at first apparently so different, are closely connected with the fungus of the first form. The foundation of these bodies consists of one or more large mother-cells filled with a mass of daughter-cells. These are clothed externally with a radiating growth, assuming a vast variety of forms. The structure often so exactly simulates that of minute moulds, that it is very difficult to get rid of the notion that they are really vegetable growths. Pure sulphuric ether, however, dissolves them completely, and shows that they are merely different forms assumed by stearine. Sometimes the white mass consists of straight slender threads radiating in every direction, each of which is surmounted by a globose or elliptic spore-like body, while occasionally the threads or crystals are shorter and the globe irregular. Sometimes the globules are absent, and in one case the fundamental cell budded like the receptacle of an *Aspergillus*, each new cell being separated by an articulation and supported on a short stalk.

Sometimes the outer coat consists of regularly dichotomous or trichotomous fascicles of linear crystals, which are free above. Sometimes, on the contrary, the fascicles are dilated above with ciliary processes, or labiated; while occasionally there are straight radiating bodies surmounted by a globular mass, pierced and surrounded by cilia. Another form appears under the guise of little feathers; while a not unfrequent one consists of leaf-like, oblong, strongly acuminate scales, simulating the leaves of mosses. *The foundation is, however, in every case, an organized cell, the red color of whose daughter-cells is precisely that of the oidium-like threads of the black fungus.* Whatever may be thought of the second and third forms of vegetable growth, this at least must be considered as identical with the first, though at present the *Chionyphe* has not been propagated from its globules—so closely involved in stearine that their germination is scarcely probable. There is not the slightest ground for supposing that the disease depends on inoculation with the spores of any of the truly parasitic fungi belonging to the tribe of *rusts* and *mildews*, but great reason, on the contrary, for looking to the origin of the fungus of the foot amongst the *mucors*, even were there not something like direct proof. It is well known that *mucedinous fungi* make their appearance within cavities of vegetables which have no apparent connection with the outward air. Nothing, for example, is more common than to find a pink mould (*Trichathrium roseum*) in the middle of a nut, and an allied vegetable production (*Dactylium soyerium*) has been found in an unbroken egg. Even the cells of plants themselves produce fungi which fructify

within them. How the spores are carried there is at present a mystery, which may some day be cleared up, as the origin of many

FIG. 59.*



intestinal worms has been, which can no longer be brought forward as an argument for equivocal generation.

[The fungi, the sole cause of the disease, are described by Dr. Carter, in his more recent publication on the subject, as follows :

(1.) The most common variety consists of small masses, of cheesy consistence and light-brown tint, formed of an aggregation of granular particles, and occupying the "loculi"—branching tubular canals passing off from the spherical cavities hollowed out in the osseous cancellous tissue. The granules or particles are visible to the naked eye, and resemble poppy-seeds; their number is immense, and they are freely discharged by the sinuses. Each consists of minute, rounded, or angular bodies (diameter about $\frac{1}{60}$ th in.), which are enveloped on all sides by a deep crystalline fringe (stearic?), and which appear to be structureless, or only finely granular; they are degenerated fungi, and in their interior may sometimes be seen clear nucleus-like forms, which somewhat resemble spores, but which are probably oil globules.

(2.) The black fungus occurs in more or less spherical masses, attaining the size of half an inch in diameter; outer surface of a jet-black color,

* Fundamental cells of the *Chionomyces Carteri* developed from the fungus foot of India, budding like the receptacle of an *Aspergillus* (BERKELEY and CARTER).

and minutely tuberculated; section of a rich deep-brown, and radiated in aspect; consistence very firm, friable, and readily yielding along the radii, sometimes tearing like decayed wood. Structure of closely aggregated fasciculi (diameter $\frac{1}{300}$ th to $\frac{1}{200}$ th in.), cylindrical, beaded, branching and blending, and radiating from a common centre; they are composed of pale, homogeneous fibres (diameter $\frac{1}{1000}$ th to $\frac{1}{3000}$ th in.), and at their peripheral extremities expand into firm, rounded "heads" of a deep black color, to the varying projection of which the tuberculated character of the exterior is due. These globular expansions (diameter $\frac{1}{100}$ th to $\frac{1}{120}$ th in.) are also found at the end of the smaller branches, and are composed of closely packed cells (beaded cellular filaments?) of an orange tint, interspersed amongst which are larger, thick-walled cells (abortive sporangia?). These larger masses occupy the "loculi," and seem to break up into smaller fragments; each of which corresponds to one or more of the globular heads thus become detached; and these black particles, incalculable in number, crowd the canals or sinuses on their way to the outer part of the body; they are larger than a pin's head, and may alone be present in the foot. Containing the reproductive elements, these black particles will, under favorable circumstances, germinate, and a red mould-like fungus springs up; this is probably the parent, or normal form of the black fungus of mycetoma. The latter also occurs in another condition, having undergone degeneration (fatty) in the foot, leaving lighter colored masses, crystalline in consistence (stearine or margarine?), and structureless; this change is an approximation to the first variety.

(3.) The countless minute pink-colored particles once seen by Dr. Carter, and referred to on p. 922, visible to the unaided eye as reddish grains (like Cayenne pepper), when examined under the microscope, exhibit a bi- or multipartite arrangement; when single they are oval, and resemble, more or less, the bodies described in the first variety, but possess the property of multiplying themselves by subdivision, and their color is different; their cellular structure is not apparent, but may have existed. The crystalline envelope is absent, though, as in the other varieties, there is much free fat of a pink tinge.

Some experiments made by Dr. Carter indicate a common origin of the two most common forms of the fungi.]

The fungus foot is confined to the natives of India, who go about with naked feet, and the spores might easily be introduced through some scratch, even were it impossible for them to penetrate by the pores of the skin. When once introduced beneath the cuticle, a single spore might soon perform the work of destruction, spreading in every direction, and, according to the peculiar condition of the secretions, the *mycelium* might put on a hundred different modes of growth. Besides, if the fungus is capable of causing the absorption of solid structures like bone, it is easy to conceive that a spore, in contact for some time with a moist foot, might penetrate the cuticle simply by absorption. Cleanliness, in the first instance, seems to be a preventive; but when the fungus is once established, there seems to be no cure save amputation—which, happily, when resorted to in time, appears to be completely successful, as the disease never spreads beyond a certain point, though, if it be allowed to take its course, death will ensue from the exhaustion consequent on pain and the continuous discharge.

[Dr. Carter (*loc. cit.*) states that he had received word (1863) from an intelligent medical man, that he thought he had eradicated the disease by the free use of strong nitric acid.]

In some cases it would seem as if the foot had been in a diseased state when the fungus was introduced; at least, the history of one case, which apparently commenced with a boil on the instep (which was treated by native doctors, a thorn being used several times as a lancet), indicates a lesion such as might well encourage the growth of a fungus parasite.

It is more than twenty years since surgeons in India first took notice of this affection of the foot in their official reports; and one of the earliest to notice the disease was Dr. Colebrook, of Madras, then Zillah-surgeon at Madura, where the endemic character of the malady was first recognized by the term "Madura foot." An interesting account of the disease was afterwards published by my friend Dr. G. R. Ballingall, who was the first to describe the microscopic peculiarities of the disease, and he was led at once to distinguish at sight the tumor of the foot from any simple scrofulous affection, and to detect the prominent features by which he recognized the fungus foot as something *sui generis*. "Cases of diseased foot," peculiar to certain parts of the Bombay Presidency, were recorded by Assistant-Surgeon Bazunjee Rustomjee, in the fifth vol. (N. S.), p. 230, of the *Transactions of the Medical and Physical Society of Bombay*, and which Dr. Carter considers are "the most fully and carefully recorded instances of the 'fungus disease'" which had been published at the time Dr. Carter wrote his report. Most of the cases belong to the second form of fungus described in the text; and practically the disease is regarded in India as a species of *caries*.

[Some idea of the frequency of the disease may be gained from the fact that individual observers in India reckon their cases by the score. Dr. Carter says that one person sent him particulars of seventy-five cases he had treated, and that even in Bombay, where the disease is not endemic, a year seldom passes without three or four cases being seen at the hospital. Other noteworthy features are: it has mostly a single local manifestation; it is much more frequent in men, and during the middle periods of life, and commonest amongst the agricultural class; it is not hereditary, nor peculiar to any diathesis (CARTER).]

Symptoms.—In the first variety the general form of the foot is oval, being much enlarged about the ankle and over the instep. On either side of the ankle-joint, on the dorsum of the foot near the toes, likewise on the sole, are numerous small soft swellings or tubercles, as large as a pea or marble, having pouting, puckered apertures, leading to fistulous canals; and the skin surrounding these apertures appears lighter in color than elsewhere. (See specimens in the Museum of the Army Medical School at Netley.) The canals sometimes lead directly to the bone; and a discolored glairy fluid, which exudes from the canal, sometimes carries with it a few black, gritty particles. The toes are distorted and displaced upwards,

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spots of one-third to one-half inch, or more, in diameter. The superficial dark layer of cuticle is cast off, leaving a very regular circular white surface, the centre of which presents a depression, closed at the bottom by a brownish layer, very thin in the middle. It was found on section that a small cavity existed beneath this depressed spot, or a tubular prolongation was detected running down through the remaining thickened cuticle and cutis into the subcutaneous cellular tissue, where it was not difficult to find the fungus particles, pink or yellowish-colored, and also in the cavity above named, in the superficial part of the cutis, or even on the surface of the latter, —the cuticle being raised.

Another and smaller specimen most clearly showed the development of the *fungi* at the very spot where, in all probability, their germs were first produced. In other parts there is a prolongation of the growth into the subjacent tissues, and there pink-colored particles were to be seen. The local nature of the whole affection—its very beginning—was here unmistakably displayed; and the superficial appearance of the skin gave the impression that a vesicle or blister had once existed there, not at all unlike that left after a *Guinea worm* has begun to discharge, as it is well known that the end of the worm makes its appearance in the centre of such a circular spot.

A further examination of the fungus particles showed their perfect resemblance to those of older specimens, and bodies not unlike spores were occasionally seen.

[It may be regarded as certain, that the hand or foot becomes accidentally inoculated with the spores of some fungus, which, at certain periods of the year, most likely during the wet season, makes its appearance on the soil of particular localities. The naked, unwashed feet of the agricultural laborer must be peculiarly liable to receive it, and the pre-existence of an abrasion of the skin is not necessary, for the spores are abundantly capable of passing into natural apertures—*e. g.* the sweat-ducts. The pinkish streaks on examination have been found to contain numerous spore-like cells in various states of growth, and probably constitute the first stage of the disease (CARTER).]

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